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Mitral Stenosis and Pregnancy.*

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INTRODUCTION.

THERE is a large amount of literature upon this subject. Many of the papers contain accounts of small numbers of cases only. References are given at the end of this article.

Berthiot's (3) book, published in 1876, and Macdonald's (11), published in 1878, have long been the standard works upon the subject. More recent publications which go fully into the question are those of Handfield-Jones (8) and Allyn (1), in 1896; Jess (9), who has collected all the published material upon the subject up to 1898; and Nicholson (13) and Mackenzie (12) in 1904.

There are certain points in regard to valvular heart disease and pregnancy upon which there is general agreement. These we do not propose to discuss further, because they appear to be well established. They are the following:—

(1) Of all the varieties of chronic valvular heart disease, mitral stenosis is that most commonly accompanied by heart failure during pregnancy.

(2) Aortic lesions without mitral are rare in women; few cases of pregnancy in women who have aortic without mitral disease come under observation.

(3) When symptoms of heart failure have preceded pregnancy they are made worse by pregnancy.

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(4) Repeated pregnancies at short intervals cause greater risk of heart failure than do few pregnancies at longer intervals.

There are, on the other hand, some points upon which there is not the same agreement. Among these, one of the most important, perhaps, is the question of whether a young woman with mitral stenosis should marry. It is to this question in particular that we devote our attention in the present paper.

The Views of Others.

The serious view that has been taken of the prognosis in patients with mitral stenosis who become pregnant is shown by the following quotations:—

Jellett, in his "Manual of Midwifery," 1905, p. 591, says:—"Finally, the question must be answered, Should a woman with valvular disease marry? The answer to the friends or relatives of the patient must be 'No.' Our advice will probably not be taken, but, all the same, it should be given, and none the less definitely on that account. There is no use in 'hedging' by saying that if failure of compensation has ever occurred, or if the damage to the valve is considerable, or if some particular valve is affected, she should not marry. In view of the sequence of events which we know to be usual in any case of valvular lesion, and remembering that a woman has duties as a wife and as a mother which require her health and strength for their due performance, there should be no hesitation in the mind of the physician as to what answer he would give to such an inquiry. It is astonishing how frequently the question is raised in text-books and how evasively it is answered. That 'the perils of marriage should be clearly stated to both the contracting parties,' as advised in a very recent American treatise on 'The Heart,' is not the way out of the difficulty. The physician has many puzzling questions to answer, but this is not one of them, and, as his opinion has been asked, it should be given in a definite and unequivocal manner."

P. Brouardel (21), quoting Porak, confirms the axiom, "Pour une cardiopathe, jeune fille, pas de mariage; mariée, pas de grossesse."

These opinions are based upon the following statistics:—

Macdonald's figures:

	No. of Cases.	No. of Deaths.	Maternal Mortality.
Mitral stenosis	14	9	64·4 per cent.
Mitral regurgitation	8	3	37 „
Aortic regurgitation	5	2	40 „

Porak's figures:

	Premature Births.	Maternal Mortality.
Aortic lesions	25 per cent.	23 per cent.
Mitral regurgitation	50 „	13 „
Mitral stenosis	30 „	61 „
Mitral stenosis and regurgitation	42 „	45 „
Complex lesions	43 „	50 „

We would point out, however, that these statistics are based upon what are virtually selected cases. They only cover those in whom the cardiac symptoms had led the patients to seek medical advice. They do not include the patients in whom pregnancy produced little or no heart failure.

This is a very important omission. We have not been able to find an analysis of any large number of cases of women suffering from mitral stenosis in which this source of fallacy has been taken into account.

We have, therefore, analysed the obstetric histories of 300 consecutive cases of mitral stenosis in women over twenty, who have been in Guy's Hospital.

We realize that it is extremely difficult to be certain of the date at which a grown-up woman with valvular heart disease first acquired it. In many cases of mitral stenosis there is no history of acute rheumatism or chorea. The mitral stenosis may be proved by autopsy to be old. It is believed that such cases have had endocarditis in childhood, when the joint pains have been so slight that they have escaped the attention of the parents.[1]

Even when there has been an attack of rheumatic fever in early youth there is often no means of determining with certainty that the valvular disease has dated from it. In our analysis we have excluded all cases where granular kidney was possible, and also those cases where the patient stated that rheumatic fever first occurred after twenty years of age. We have taken those in which the clinical diagnosis has been old-standing mitral stenosis, with or without other lesions, and in which there has been either rheumatic fever or chorea in childhood or youth, or no history of acute rheumatism at all. We have accepted the same evidence in all the cases, whether in married

[1] Taylor, in 'The Practice of Medicine,' 1904, p. 157, says: ". . . the cardiac lesions may occur without any obvious affection of the joints at all. This greater liability on the part of the heart is especially frequent in children. . . ."

women not pregnant, in married women pregnant, or in single women over twenty, so that the analyses of each class are comparable. Our cases are given in tables at the end of the paper.

Many Mitral Stenosis Patients bear Children well.

The likelihood is, that any woman who has mitral stenosis will, sooner or later, suffer from the results of failing compensation. There are all degrees of mitral stenosis, and of the power of different hearts to maintain their compensation. Some hearts will fail early, whatever the woman does. Other hearts seem able to carry on their work almost as well as if no valvular disease were present. Even when heart failure comes on during pregnancy or the puerperium it is difficult to be sure that the heart would not have failed in any case, even had there been no pregnancy.

We have analysed over 300 cases as justly as we are able, attributing heart failure to child-bearing in as many as we felt we honestly could. We have come to the conclusion that the greater number of pregnancies in women with mitral stenosis, whose compensation has not previously failed, run their course as naturally as do the pregnancies of healthy people.

Thus, of the 300 consecutive cases, 205 were married. Of these, 135, or 66 per cent., did not attribute their ultimate heart failure to pregnancy, nor could we satisfy ourselves that there was any direct relation between the pregnancy and the heart failure. In one of these cases there had been as many as 17 children born alive, and the average number of children was 4·5 per mother. If 135 mothers with mitral stenosis can bear 608 children without losing cardiac compensation, it would seem unjust to prevent a young woman with compensated valvular heart-disease from getting married.

We found a direct relation between child-bearing and heart failure in 57 women, or 28 per cent. In many of these, however, there had been previous children born without trouble. In one case, indeed (No. 168), the labours with twelve children had been uneventful, heart failure occurring for the first time with the thirteenth. Upon twelve separate occasions this case might have come into our group of cases without heart symptoms; but the thirteenth transfers her to our group of cases where heart failure is related to pregnancy. It seems worth while to represent the relationship between pregnancy and heart failure in mitral stenosis in another way, as follows:—

	Associated with Heart Failure.	Not Associated with Heart Failure.
1st pregnancy	15	177
2nd „	16	139
3rd „	10	116
4th „	14	95
5th „	13	74
6th „	14	61
7th „	5	50
8th „	8	38
9th „	1	30
10th „	2	26
11th „	2	18
12th „	2	13
13th „	2	10
14th „	0	7
15th „	0	2
16th „	0	2
17th „	0	1

The Time at which, when related to Pregnancy, Heart Failure sets in.

We appreciate fully the fact that an existing tendency to failure of compensation is aggravated by child-bearing. Some of these patients, when they do go wrong, break down badly. Others, however, respond no less readily to treatment than do non-pregnant cases. It is difficult to determine the prognosis in any given case.

Amongst the 57 patients (see Table, Nos. 149—192) in whom we relate the cardiac failure to child-bearing we were uncertain in 7 whether the symptoms came on before, during, or after the birth of the child. In the remaining 50, 25 dated their heart trouble to the time when they were pregnant, 25 went to term without difficulty, and the cardiac symptom set in during the puerperium.

The Prognosis when Heart Failure is Related to Pregnancy, Labour, or the Puerperium.

The prognosis in regard to heart cases is always difficult to estimate from hospital records. Many patients recover sufficiently to go away to their homes, but there is no evidence to show how long their cardiac compensation is maintained. Some such cases doubtless die comparatively soon. Others remain chronic invalids for years. A few recover sufficiently to do their work for a longer or shorter time. It is a matter of every-day experience to find heart cases, men and women alike, coming into hospital for a few weeks, recovering cardiac compensation to some extent, going away to their homes only

to return again and again to the hospital. Those who die at home are not heard of again. Those who recover completely for the time being are also lost sight of. They change their address and cannot be traced. There is the greatest difficulty, therefore, in determining whether women with mitral stenosis, whose cardiac compensation has broken down in relation to child-bearing, have a worse prospect of life than have other patients whose heart failure has been due to other causes.

The proportion who die in the hospital is really no criterion, because we do not know what proportion of the others die soon after discharge; but since this source of error is common to all hospital statistics, we give the proportions for what they are worth:—

(*a*) Of 135 mitral stenosis patients who had borne children, but whose heart failure did not date from child-bearing, 44, or 33 per cent. died in hospital.

(*b*) Of 57 mitral stenosis patients who had borne children, and whose heart failure did date from child-bearing, 20, or 35 per cent., died in hospital.

(*c*) Of 13 mitral stenosis patients, married but never pregnant, 6, or 46 per cent., died in hospital.

(*d*) Of 95 mitral stenosis patients, unmarried, 17, or 18 per cent., died in hospital.

At first sight this would seem to indicate that the prognosis was worst in the sterile women, best in the unmarried, intermediate in those who had families. A glance at the relative ages in the different groups shows that this deduction is unwarranted; for the average age of all the cases in the four groups was:—

	Average Age.[1]	Maximum Age.	Minimum Age.
(*a*)	41 years ...	71 ...	22
(*b*)	32 „ ...	48 ...	20
(*c*)	34 „ ...	55 ...	25
(*d*)	30 „ ...	60 ...	20

The average age of the single women was less than that of the married; the mortality amongst them should naturally be less. Could we trace the unmarried patients forward into the ten years to come, we should find that many would ultimately die in hospital, and some of these would probably have entered into the married

[1] The average age at death of married women with mitral stenosis is obviously less than that of healthy women. If the fact that the wife is likely to predecease the husband is regarded as a bar to marriage in all cases, then we agree that women with mitral stenosis should not marry. Our point is that we think the grave influence of pregnancy upon mitral stenosis has been over-estimated.

state before they died. Many of our married patients had come in and out of hospital half a dozen times or more before they ultimately died.

We think the hospital mortality statistics afford no sound basis for any deduction; but if we drew any deduction at all it would be that, allowing for differences of age, the mortality of matrons with mitral stenosis is not materially different from that of spinsters with mitral stenosis.

The Prognosis when Heart Failure sets in during Pregnancy.

The paragraph above indicates how difficult it is to say whether or not a given woman, a hospital patient suffering from mitral stenosis, with symptoms of heart failure, will ultimately die in hospital or not. It is less difficult to say whether or not a given woman, being pregnant, and admitted to hospital with cardiac symptoms from mitral stenosis, will leave the hospital alive, and whether or not she will approximately reach term and bear a living child.

Amongst over 300 consecutive cases, 22 were admitted whilst actually pregnant. For the details of these we refer to the table at the end of the paper. In addition to these, we have found fourteen other pregnant mitral stenosis patients, who came into the hospital either before or after the period of our 300 consecutive cases. The following are notes of these additional patients:—

(i) Aged 43. She was admitted for retroverted gravid uterus, and had no cardiac symptoms; there was well-marked mitral stenosis. The uterus was replaced, the patient being in the ward only five days. She had been married fifteen years, and had had seven living children and one miscarriage. The last labour was seventeen months before, at full term. She was now pregnant four months.

(ii) Aged 36. She was admitted when seven months pregnant for orthopnœa, precordial pain, hæmoptysis, and bronchitis, without œdema. She gave no history of acute rheumatism, but was found to have old mitral stenosis. With rest in bed and digitalis she improved rapidly. She went to term. The labour was natural. She and her child both did well. She had had ten living children previously, and with each pregnancy had had some dyspnœa in the later months, but had recovered completely soon after labour.

(iii) Aged 22. She was admitted when eight months pregnant for her eleventh attack of acute rheumatism. She had mitral stenosis and regurgitation, and aortic stenosis and regurgitation, but neither

now nor previously had she suffered from her heart. She went to term; labour was natural; mother and child did well. She had had one child previously, stillborn at full term, without difficulty. She had been in Guy's Hospital eleven times before, once for hæmatemesis and (?) gastric ulcer, ten times for acute rheumatism. The heart lesion was old.

(iv) Aged 40. She had been married only six months, and was five months pregnant on admission. She came in for dyspnœa. She rested in bed for a fortnight, and went out on the twenty-fourth day, free from dyspnœa, still pregnant. The heart lesion was old mitral stenosis.

(v) Aged 25. She came in for dyspnœa when four months pregnant, and was found to have a large, irregular heart, and mitral stenosis and regurgitation. She was only in the ward six days, when she went home of her own accord, still pregnant. She had had rheumatic fever four times.

(vi) Aged 19. She came in when pregnant nearly to term for a sudden hemiplegia. This was found to be due to cerebral embolism from mitral stenosis. There were no cardiac symptoms. She went to term. Labour was natural. Mother and child did well, but the hemiplegia passed off but partially. There was weakness of the affected side a year later, but no heart failure. There was no history of rheumatic fever.

(vii) Aged 33. She came in for acute bronchitis and orthopnœa, without œdema, when six months pregnant. She was found to have mitral stenosis, but gave no history of acute rheumatism. She was immediately relieved by rest in bed, and went out in fifteen days, still pregnant. She had had some trouble with her first pregnancy, but had recovered completely, and had borne seven living children.

(viii) Aged 20. She had had acute rheumatism many times, first when eleven. She had aortic stenosis and regurgitation, and mitral stenosis and regurgitation. She had had one living child two years before without difficulty, and had now missed two menstrual periods. Until just before admission she had worked hard at a jam factory, carrying trays of jars of jam up and down stairs. She was seized with acute rheumatism again, and came to hospital with a certain amount of dyspnœa also. She rested in bed, recovered rapidly, and went out on the twentieth day, able to walk actively without dyspnœa. It was jam-jar carrying rather than pregnancy that had caused the cardiac symptoms.

(ix) Aged 29. She gave no history of acute rheumatism, but had old mitral stenosis. She had had four children previously without

difficulty. Eighteen days before admission orthopnœa and cough came on simultaneously with an abortion. She was attended in the out-door maternity department and transferred to the wards. She rested, and had digitalis; on the twenty-sixth day she went out, free from dyspnœa.

(x) Aged 25. She had had acute rheumatism at sixteen and at twenty-one. She came in for dyspnœa in the later months of pregnancy, and was found to have mitral disease. The notes are incomplete; it is not known if she was married or if she had had a previous pregnancy. With rest and digitalis she became free from dyspnœa, and went out on the twenty-fourth day, still pregnant.

(xi) Aged 27. She had had no acute rheumatism, but had old mitral stenosis. She had been married four years. Her first pregnancy ended at the seventh month in the delivery of a still-born child. The second pregnancy went to term naturally, and there was no heart failure, but when two and a half months pregnant she had a "fit," which left her with hemiplegia. This passed off completely after labour. Dyspnœa first began fourteen months ago, and on admission she was eight and a half months pregnant, orthopnœic, and cyanosed. With rest in bed and digitalis she reached full term, and was delivered of a living female child weighing 6 lb. 8 oz. She and her child did well, and she went out early in the puerperium. The dyspnœa was still present on exertion, but not with ordinary walking.

This patient became pregnant again a year and a half later. She was admitted at the fourth month for hæmatemesis, and rapidly recovered from this, but all through the pregnancy there was severe dyspnœa with swelling of the feet. Cyanosis became extreme, and just before term labour was induced. Unassisted delivery took place twenty-four hours later, and was accompanied by post partum hæmorrhage. The child was 17 inches long, weighed 6 lb. 8 oz., and lived. The mother had severe dyspnœa and bronchitis during the early part of the puerperium, but under treatment the œdema disappeared and the cough decreased. She walked from the hospital, but readily became dyspnœic on exertion.

(xii) Aged 22. She gave no history of acute rheumatism, but was found to have mitral stenosis. She did not come in for heart failure in the ordinary sense, but for acute pericarditis. She refused to stay in the hospital. On the third day she insisted on going home, notwithstanding that she had acute pericarditis and was very seriously ill. She was pregnant five months at this time, and had borne one child eighteen months previously without developing cardiac symptoms.

(xiii) Aged 26. She gave no history of acute rheumatism, but was found after death to have chronic valvular heart disease, both aortic and mitral, and a fatty heart. She had been married a year, and was pregnant nearly to term. She had developed acute dyspnœa three weeks before. Labour was induced and a living male child born. The patient became much worse the day after the confinement, and the heart did not respond to any treatment. The mother died on the ninth day after labour, the child lived.

(xiv) Aged 24. She gave no history of acute rheumatism, but had mitral stenosis. She had had twins prematurely thirteen months before. The infants were born living, but both died. There had been no cardiac symptoms with that pregnancy. When five months pregnant for the second time she became very dyspnœic and cyanosed. When admitted, it was thought she must die; she recovered rapidly with rest in bed and digitalis, and was able to go home, still pregnant. She was re-admitted at the seventh month, extremely dyspnœic, with œdematous legs and a rapid, irregular pulse. She was bled, and digitalis was given, and she rested in bed. The pregnancy continued naturally; the cardiac symptoms all abated; she was delivered at full term of a living child weighing 5 lb. 6 oz. Both mother and child did well, and the mother was free from dyspnœa on ordinary exertion when she left the hospital.

We have, therefore, 36 cases in which mitral stenosis patients have come into Guy's Hospital when pregnant. These are all we have been able to find in a period of over twenty-five years. Leaving out patients under twenty years of age, the number of women with mitral stenosis who were admitted during the same period was something like 750. If cardiac symptoms from mitral stenosis were the rule during pregnancy, surely more patients would have sought admission when actually pregnant.

Of the 36 patients, not one died during pregnancy, if we exclude Cases Nos. 149 and xii, who refused to stay in and whose fate is not known. Not one died during labour. Nine had no heart failure, but came in for other things (Nos. 4, 5, 8, 165, 168, i, iii, vi, xii). Twenty-four went out with restored cardiac compensation (Nos. 4, 5, 8, 151, 152, 153, 155, 161, 163, 165, 166, 168, 169, 171, i, ii, iii, iv, vi, vii, viii, ix, x, xiv). Only five died within three months after labour (Nos. 174, 177, 180, 183, xiii), and of these one (No. 180) died, not of mitral stenosis, but of chorea gravis and infective endocarditis.

In regard to the children, the fate of ten is unknown, because the mothers recovered and went out to be delivered elsewhere. Of the remaining 27, 23, including twins in one case, were born living, at

term, or within a month of term (Nos. 4, 5, 8, 151, 155, 161, 163, 165, 166 (twins), 169, 174, 177, 178, 182, 183, ii, iii, vi, xi (?), xiii, xiv). In two cases (Nos. 153, 171) the child was born at or near term, but dead. There were two abortions (Nos. 180, ix), and the former of these was due to chorea gravis.

These figures are very different from those of Macdonald (11), as will be seen by comparing them side by side:—

	No. of Cases.	Maternal Mortality within Three Months.	Abortions.	Lesion.
MACDONALD: (Published cases)	14	64·4 per cent.	14·3 per cent.	Chronic mitral stenosis only.
OURSELVES: (Consecutive hospital cases)	36	13·9 „	5·5 „	Chronic mitral stenosis, with or without other lesions.

We very much wish we had a larger number of cases in which the course of pregnancy in mitral stenosis had actually been observed in hospital. We feel that the great difference between Macdonald's statistics and our own is in part due to the small number of cases we each have. Nevertheless, we feel convinced that Macdonald's figures overstate the seriousness of the prognosis. His own words are:— "We have thus nine cases out of fourteen, or 64·4 per cent., fatal, which indicates a tendency to death which is surely sufficiently grave. It will be observed that the deaths occurred either suddenly during the labour or within a few days or weeks afterwards." We agree that the cardiac failure, once begun, may become very grave during the puerperium, but we have no single instance in which death occurred during labour.

The patients behave very much like other cases of heart disease. Even when the heart condition seems hopeless they may recover and bear other children. An instance in point is No. 169, whose history was shortly as follows:—

She became dyspnœic during her first pregnancy, and had had cardiac trouble many times since. On two separate occasions her symptoms were so grave that labour was induced at the eighth month; on one of these there was post partum hæmorrhage, which nearly proved fatal. After her fourth child she was discharged from the hospital, with the note in her report that she was "a wreck"; at that time it was thought impossible that she could live, but she recovered

at home, and bore two more children. The last, and sixth, was born at term, without induction of labour; it was a transverse presentation and version had to be performed; the mother and child both did well.

The Treatment of Mitral Stenosis Patients when Pregnant.

The patients with mitral stenosis who have come into Guy's Hospital pregnant have, almost without exception, been treated as though they were not pregnant. Rest in bed, with digitalis, given with the same precautions as in other cases, has almost invariably brought relief, and enabled the patient to go on to natural labour at or near term. Induction of labour has hardly ever been resorted to, as reference to the cases at the end of this paper shows. Labours have in almost all cases been easy and natural, and free from post partum hæmorrhage.

It is true that the same might not hold good for ladies in higher ranks of life. The physical work of women living in the "Borough" is hard, that of most well-to-do women is less so. The relief to the "Borough" woman's heart is proportionately greater than is that to the rich lady's when she goes to bed. Nevertheless, we hold the view that the treatment of a pregnant woman with mitral stenosis should not be different from that of a non-pregnant woman with the same heart lesion. If the patient can be up and about, without cardiac symptoms, it is better for her to live as usual, and by moderate exercise maintain the reserve power of her heart, rather than lie up and diminish this reserve power by prolonged rest. If cardiac symptoms supervene, the treatment should then be rest on a couch for mild cases, rest in bed for severer cases, rest in bed and digitalis for severer still. The pregnancy should, if possible, be allowed to run its course. Induction of labour in cardiac cases brings no immediate abatement of symptoms, as it does in many cases of eclampsia, for example. The puerperium is not less dangerous than is pregnancy itself to a patient with mitral stenosis. The cardiac condition should be restored to as fair a state of compensation as possible before the time of labour arrives, and then forceps may be used to assist Nature. In a word, treat the patients exactly as though they were non-pregnant; treat them for mitral stenosis, do not treat them for pregnancy.

Sterility in Mitral Stenosis.

The opinion has been expressed that many women with mitral stenosis are sterile. Allyn (1), for example, says that "mitral disease, particularly stenosis, is much graver, as a rule, than aortic, but

there is an attempt at a natural prevention of this, owing to the high proportion of sterile women among the subjects of mitral stenosis."

We do not agree with this. Out of the 205 married women in our table, only thirteen had not been pregnant. One of these had but recently got married, so that the proportion of presumably sterile women was only 5·8 per cent. The remainder had borne, upon the average, between four and five children apiece.

The Liability to Abortion in Mitral Stenosis.

Allyn (1), quoting Porak (22), states that cardiac disease in the mother has a very grave influence upon the fœtus, abortion being very common.

Unfortunately, this point was not particularly attended to in many of our cases. In our epitomes we have only put down whether abortions had occurred or not when we had definite statements from the patient to that effect. We have left the doubtful cases blank.

In 90 of the women who had been pregnant we ascertained the history in regard to abortions, and found that 40 of them had never had any abortion at all. The remainder had had 91 abortions between them. The general average was thus 1 per mother. The majority did not tend to abort, but in a few there were repeated abortions—in Case No. 56 as many as six.

It will be noticed that some of the abortions occurred when there was no heart failure at all. In these the association was possibly adventitious. In others the heart failure dated from an abortion, and it seems likely that in some of these the heart trouble was directly responsible for the miscarriage.

Upon the whole, however, we do not think that the tendency to abortion is obviously greater amongst mitral stenosis patients than it is amongst other "Borough" women.

Cases in which we know that the Mitral Stenosis certainly Antedated the Pregnancies.

As we have pointed out in the early part of this paper, it is impossible to state with absolute certainty in a large number of cases that the mitral stenosis was present before marriage. We have said that this is a flaw in our arguments, and might render the deductions we have drawn from our 300 cases invalid. There is, however, a small number who had been in the hospital, or under observation, years previously, and in whom we know that mitral stenosis was pre-

sent before marriage. We will now consider these, seventeen in number, by themselves, and see whether what we have said about the generality of the cases holds good of these also.

Case No. 6.—Valvular disease was known to exist at ten. There had been one child, and there had never been cardiac symptoms. The patient was admitted for a fourth attack of acute rheumatism, with good cardiac compensation.

Case No. 12.—There had been acute pericarditis before marriage. There had been one living child and one miscarriage. The patient was admitted for recent cardiac symptoms, not related to child-bearing.

Case No. 59.—The physical signs of mitral disease had been present for thirty years. The patient had borne ten children without difficulty. Heart failure did not set in till she was fifty-six.

Case No. 75.—The mitral bruits were present at twelve. The patient had had five children. She came in for acute rheumatism, and had never had cardiac failure.

Case No. 89.—Heart disease was known to exist at fourteen. There had been one child, without difficulty. The patient came in for lobar pneumonia, and recovered without a symptom of heart failure.

Case No. 90.—The bruits were known to exist before marriage. There had been three children, born without difficulty.

Case No. 91.—The bruits were known to exist before marriage. The patient bore five children, and her heart failure did not come on in relation to any of these.

Case No. 92.—Heart disease was known to exist at thirteen. There had been three children, pregnancies and labours being uneventful.

Case No. 93.—Heart disease was known to exist at thirteen. There had been four children and two miscarriages, without trouble.

Case No. 94.—Heart disease was known to exist at sixteen. The four children had been born without cardiac symptoms.

Case No. 95.—Heart disease had been known to exist for ten years. There had been eight children, and no heart failure with any of them.

Case No. 96.—Heart disease was known to exist in girlhood. There had been one child, born without trouble.

Case No. 97.—The bruits were known to be present at nineteen. There had been one child, born without trouble.

Case No. 138.—Heart disease was known to exist at sixteen. There had been six children. Cough and dyspnœa had occurred during each pregnancy, but there had been good recovery of compensation each time.

Case No. 148.—Heart disease was known to exist at sixteen. The first five children had caused no cardiac symptoms. Failure of compensation set in with the sixth.

Case No. 153.—This patient had been in and out of hospital seven times for heart failure before marriage. She married notwithstanding. The cardiac symptoms were severe during pregnancy. A dead child was born at the eighth month. The mother recovered rapidly enough to leave the hospital on the fourteenth day after labour.

Case No. 161.—This patient was in hospital when eighteen for heart disease. She married after this, and bore four children without heart trouble. When pregnant with her fifth child, cardiac symptoms appeared. The patient lay up in hospital for four days only, and then went home and went naturally to term.

There were, it will be seen, many children borne by women who were known to have heart disease before marriage. In 13, or 76·5 per cent., the ultimate heart failure was not directly related to child-bearing. In 4, or 23·5 per cent., pregnancy and heart failure co-incided, but even in some of these previous children had been born without causing heart trouble. None of the patients died during pregnancy or labour. All recovered and left the hospital.

If we compare these figures with those for the generality of women with mitral stenosis, we find:—

	Heart Failure not directly related to Pregnancy.	Heart Failure directly related to a pregnancy not necessarily the first.
When the mitral stenosis was old, but of unknown date (175 cases, taken consecutively)	69·7 per cent.	30·3 per cent.
When the mitral stenosis was known with certainty to date from before marriage (17 cases, taken consecutively)	76·5 „	23·5 „

The results are closely similar. We are fully conscious that the number of cases in which we know that the mitral stenosis certainly preceded marriage is small. In the remainder the evidence that the mitral stenosis was present before marriage is presumptive only. We do not know how to collect a large number of cases where this presumption is avoidable. We have taken only those cases where the bruits suggested an old-standing valvular lesion, and have only accepted cases where there had either been acute rheumatism or chorea in youth or else no rheumatism at all. The fact that the results are so similar in the total number of cases to what they are in those where heart disease was known to antedate the pregnancies affords, we think, additional ground for the justness of the conclusions we have drawn.

Association of other Heart Lesions with the Mitral Stenosis.

Most observers are of the opinion that the prognosis is less good when aortic or other disease is present as well as mitral stenosis. We have taken our cases consecutively as they were admitted to the hospital, and have made no distinction between cases where mitral stenosis alone was diagnosed and those where other lesions of the heart were present also. Amongst the associated lesions will be found mitral regurgitation, aortic regurgitation, aortic stenosis, aortic stenosis and regurgitation, pulmonary stenosis, tricuspid stenosis, pericarditis, and adherent pericardium. Notes of these are given in the epitome of cases in the table at the end of this paper. They should make the prognosis in the affected cases proportionately worse. We do not intend to enter upon this question here. We have discussed the cases as though the patients were suffering from mitral stenosis only.

The Incidence of Fungating Endocarditis.

In all the patients who died the diagnosis was verified by autopsy. We have been struck by the large proportion of mitral stenosis patients who die of a terminal fungating endocarditis. Thus:

Of 43 fatal cases where failure was not dated to pregnancy, 10, or 23 per cent., of the patients died of fungating endocarditis.

Of 22 fatal cases where failure was dated to pregnancy, 9, or 41 per cent., of the patients died of fungating endocarditis.

Of 6 fatal cases in patients who were married, but had not been pregnant, 0 per cent. died of fungating endocarditis.

Of 18 fatal cases in patients who were single, 7, or 39 per cent., died of fungating endocarditis.

Of the total 89 fatal cases, 26, or 29 per cent., of the patients died of fungating endocarditis.

At first we thought there might be a special tendency for pregnancy or the puerperium to lead to fungating endocarditis, but we do not think this can really be so, seeing how high the proportion of cases of terminal fungating endocarditis is in single women with old mitral stenosis.

Summary.

We believe that heart failure is to be expected sooner or later in almost all cases of valvular heart disease.

We do not deny that pregnancy may cause serious, and even fatal, cardiac failure in cases of mitral stenosis.

We think, however, that the dangers of pregnancy in these cases have been overstated.

We attribute the overstatement to the fact that previous statistics have been based mainly upon cases of mitral stenosis which came under observation because heart failure had developed during, or soon after, pregnancy. We feel that statistics so obtained leave out of count all those patients with mitral stenosis who go through pregnancy without developing cardiac symptoms.

We have tried to obviate this source of error by analysing the obstetric histories of 300 women over twenty who had mitral stenosis with or without other lesions. We have not selected our cases, but have taken them consecutively as they came into Guy's Hospital.

We conclude:—

(1) That comparatively few are sterile.

(2) That they are not especially liable to abort.

(3) That the majority bear children well.

(4) That when heart failure develops in relation to pregnancy it is very often not with the first pregnancy, but after several pregnancies.

(5) That the treatment should be the same as for a non-pregnant patient with mitral stenosis.

(6) That it is not just absolutely to negative marriage in all women with mitral stenosis. The dogmatic "no" of Jellett and of Porak (p. 2) is, we think, unjustifiable. It is right that the physician should make clear to the contracting couple, or to their near relatives, the risk run. He should use his discretion, and distinguish between one case and another. The risk should not be minimized, but it should not be exaggerated. Whether the woman marry or not, it is

likely that she will not reach old age. She should not have successive children rapidly. But if she has survived the age of twenty, with good cardiac compensation, the likelihood that pregnancy will accelerate the time of heart failure does not seem to be so great as has been declared in text-books.

We thank the Treasurer of Guy's Hospital and the Physicians to Guy's Hospital for their kind permission to use the statistics embodied in this paper.

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Analysis of 300 Consecutive Cases of Mitral Stenosis, with Special Reference to Pregnancy and Labour. No Case is included under the Age of 20; and in all Rheumatism or Chorea had occurred before 20, or not at all.

A. *Those who had been Pregnant, and did not date Cardiac Symptoms to Pregnancy or Labour.*

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result*
1	22	None	1	0	Mitral stenosis, acute bronchitis, erythema nodosum	Œdema and dyspnœa	7 weeks	Married 18 months. One child, full term, 8½ months ago without trouble	R.
2	24	19	1	0	Mitral stenosis and regurgitation, infective endocarditis, various emboli	Pyrexia and rigors	No heart failure	The pyrexia and sepsis date from 1 month after labour; the pregnancy and labour had been free from cardiac symptoms. The infection may have been directly due to the puerperium, but there was no cardiac failure	D.
3	25	None	1	0	Lobar pneumonia (double), mitral stenosis	Pneumonia	No heart failure	The child was born at full term 12 days before admission. There were no cardiac symptoms at all	R.
4	27	,,	3	0	Chorea, mitral stenosis	Chorea	No heart failure	Two full-term children without trouble. At present 7 months pregnant; subsequently went to term without cardiac symptoms	R.
5	27	13	1	0	Chorea, mitral stenosis	Chorea	No heart failure	Patient unmarried, and 4½ months pregnant on admission. Recovered from chorea; went to term naturally	R.

* R = recovered and went home D = died in Hospital

6	27	6	1	0	Acute rheumatism (4th), mitral stenosis	Rheumatism	No heart failure	Known to have had heart disease at 10; no cardiac symptoms since	R.
7	33	10	4+1†D.	1	Acute rheumatism (2nd), mitral stenosis and regurgitation	Rheumatism	No heart failure	Last confinement was 4 years ago; miscarriage 10 weeks ago. No heart failure at labours	R.
8	36	None	8	0	Acute rheumatism (1st), old mitral stenosis	Rheumatism	No heart failure	Had had 7 children, last 2 years ago. Now admitted at term; labour natural; no cardiac failure	R.
9	25	18	3	0	Cerebral embolism, mitral stenosis and regurgitation	Hemiplegia	No heart failure	First child born at 18, second at 19, third at 28; no cardiac failure with any of them. Transient hemiplegia 14 months ago; complete, 7 months ago	R.
10	24	15	1	—*	Mitral stenosis and regurgitation, bronchitis, enlarged liver	Precordial pain and dyspnœa	1 month	The child was born without trouble 2 years ago	R.
11	28	None	3	2	Mitral stenosis, tricuspid regurgitation, œdema.	Cyanosis and dyspnœa	3 weeks	There had been no cardiac symptoms with any of the pregnancies	R.
12	30	20	1	1	Mitral stenosis and regurgitation	Dyspnœa	Acute	There had been pericarditis before marriage; the pregnancies had been uneventful	R.
13	31	16	2	—	Mitral stenosis and regurgitation, pleurisy with effusion	Dyspnœa	1 year	Pregnancies uneventful	R.
14	32	14	2	—	Mitral stenosis and regurgitation	Dyspnœa	Recent	Pregnancies uneventful	R.
15	32	None	1	—	Mitral stenosis, tricuspid regurgitation	Ascites	3 months	Child was born 7 years before	R.
16	33	Childhood	5	—	Mitral stenosis and regurgitation, tricuspid regurgitation, pleurisy	Dyspnœa and rheumatism	Recent	Last child was born 2 years before, without trouble	R.
17	34	None	1	0	Mitral stenosis and regurgitation, tricuspid regurgitation	Œdema	Recent	Child was born 5 years before	R.
18	36	None	1	—	Mitral stenosis and regurgitation	Œdema and dyspnœa	5 years off and on; acute 3 weeks	Child was born 11 years ago, without cardiac trouble	R.
19	36	,,	2	—	Mitral stenosis, aortic regurgitation	Precordial pain, angina	Acute 14 days	Pain of an anginal character had been present off and on for several years; the pregnancies had been uneventful, without heart failure or increase of pain	R.

† D = stillborn

* The mark — signifies that it is not known whether there had been any miscarriages or not

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
20	36	19	6	—	Mitral stenosis, aortic stenosis and regurgitation	Dyspnœa and œdema	18 months off and on; acute 3 months	There had been no dyspnœa except on exertion until quite lately; the pregnancies had occurred without heart failure	R.
21	36	Childhood	6	—	Mitral stenosis and regurgitation	Dyspnœa and cough	Acute recently	She had not noticed any cardiac symptoms until 3 years before; the pregnancies had been uneventful	R.
22	37	16	2+ 1 $\dagger_{12}^{7}$	2	Mitral stenosis and regurgitation	Dyspnœa	Recent	The patient stated that she had not felt thoroughly well for many years; had had no trouble with any pregnancy or labour, and had only recently felt worse than usual	R.
23	38	8	3+ 1 d.	—	Mitral stenosis and regurgitation, tricuspid regurgitation	Œdema and dyspnœa	1 year	Pregnancies uneventful	D.
24	38	Childhood	8	2	Mitral stenosis	Dyspnœa and precordial pain	On and off for 12 years; acute 1 month	Had been married 18 years. Though there had been shortness of breath on exertion for 12 years, the pregnancies had not caused any serious trouble	R.
25	38	16	2	0	Mitral stenosis	Œdema of legs	14 days	Last child was born 14 years ago	R.
26	38	None	8	—	Mitral stenosis, tricuspid regurgitation, enlarged liver	Hæmoptysis and hepatic pain	2 weeks	There had been twins twice. With each of these there had been hæmoptysis, but beyond that no heart trouble till 2 weeks ago	R.
27	39	,,	6	—	Mitral stenosis and regurgitation, enlarged liver, ascites	Œdema and ascites	2 months	Pregnancies uneventful	R.
28	39	Childhood	2	1	Mitral stenosis and regurgitation, tricuspid regurgitation, ascites	Dropsy	2 months	Pregnancies uneventful	R.

$\dagger^{7}/_{12}$=7 months child.

29	39	None	8	4	Mitral stenosis and regurgitation, pericarditis	Precordial pain	Acute	Pericarditis was the main cause for admission. No previous heart failure	R.
30	39	,,	3	—	Mitral stenosis	Dyspnœa	On and off for 4 years	Last child 12 years ago	R.
31	40	,,	13	—	Mitral stenosis, enlarged liver	Dyspnœa and œdema	On and off for 7 years; acute for 5 months	Pregnancies uneventful	R.
32	40	14	1	0	Mitral stenosis, aortic regurgitation, tricuspid regurgitation	Palpitation and œdema	12 years on and off	Child 23 years ago	R.
33	40	None	0	2	Mitral stenosis and bronchitis	Dyspnœa	2 years on and off	Miscarriages were 20 years ago with first husband. Married a second time, no children	R.
34	42	,,	2	—	Mitral stenosis, tricuspid regurgitation, bronchitis	Dyspnœa	2 years on and off	There was hemiplegia 11 years ago; the pregnancies were uneventful, and there was no cardiac failure till 2 years ago	R.
35	42	Girlhood	4	—	Mitral stenosis and regurgitation, ascites	Œdema and ascites	2½ years on and off	Last child 6 years ago	R.
36	43	None	1 + 3 d.	1	Mitral stenosis and regurgitation	Dyspnœa and precordial pain	2 years	Last child 7 years ago	R.
37	43	20	8	—	Mitral stenosis, tricuspid regurgitation	Dyspnœa and cough	8 years on and off	Last pregnancy long preceded heart symptoms	R.
38	43	3	3	3	Mitral stenosis and regurgitation	Dyspnœa and cough	14 days	Pregnancies uneventful; miscarriages without heart failure	R.
39	43	17	1	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Œdema and cough, ascites	2 years; ascites 6 weeks	The pregnancy was 25 years ago	R.
40	44	10	2	0	Mitral stenosis	Dyspnœa and precordial pain	Slight	The patient was married at 17 and quickly had 2 children	R.
41	44	9	1 + 1 d.	1	Mitral stenosis, angina pectoris	Angina and hæmoptysis	6 years	Pregnancies uneventful	R.
42	45	None	1 d.	—	Aortic stenosis and regurgitation, mitral regurgitation, pericarditis	Precordial pain and dyspnœa	2 months	Child was stillborn 20 years ago	R.
43	46	14	7	0	Mitral stenosis and regurgitation, aortic stenosis, pleurisy	Acute pleuritic pain	None	Last child 6 years ago	R.
44	47	20	8	—	Mitral stenosis and regurgitation	Bronchitis and œdema	5 months	Pregnancies uneventful; quite well till hæmoptysis 5 months ago	R.
45	47	18	4	0	Mitral stenosis and regurgitation, bronchitis	Cough	3 years on and off	Married at 18; youngest child is 25	R.
46	47	None	14	1	Mitral stenosis and regurgitation, enlarged liver	Dyspnœa and vomiting	4 days	Last pregnancy 2 years ago	R.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
47	47	11	2	—	Mitral stenosis and regurgitation, bronchitis, enlarged liver	Œdema and cough	6 weeks	Last child 20 years ago	R.
48	47	None	6	1	Mitral stenosis	Œdema and palpitation	2 months	Married at 15; pregnancies uneventful	R.
49	48	16	9	—	Mitral stenosis and regurgitation; hæmaturia	Dyspnœa	6 months	Married at 19; pregnancies uneventful	R.
50	49	None	14 + 1 D.	1	Mitral stenosis and regurgitation, ascites	Œdema	2 years	Married twice; 8 and miscarriage by first husband; 6 and 1 still-born at 7 months by second	R.
51	50	,,	11	—	Mitral stenosis and regurgitation	Palpitation and œdema	3 years on and off	Last child 7 years ago; patient has been a widow for 5 years	R.
52	50	,,	9	—	Mitral stenosis	Dyspnœa and œdema	6 years on and off	Pregnancies uneventful	R.
53	51	,,	14	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Anasarca	3 years	Pregnancies uneventful	R.
54	53	14	1	—	Mitral stenosis and regurgitation	Dyspnœa	2½ years	Child born many years before	R.
55	53	None	17	—	Mitral stenosis and regurgitation, tricuspid regurgitation, ascites	Dyspnœa and œdema	1 year	Pregnancies uneventful	R.
56	54	7	2	6	Mitral stenosis, bronchitis, extreme cyanosis	Dyspnœa	1 month	Pregnancies uneventful	R.
57	55	15	14	—	Mitral stenosis and regurgitation, bronchitis	Dyspnœa and œdema	1 year	Last child 15 years ago	D.
58	56	20	14	—	Mitral stenosis, ascites	Cough and œdema	10 weeks	Married at 20, and had her children quickly and without heart trouble	R.
59	56	15	10	—	Mitral stenosis	Dyspnœa and œdema	1 year	There was no trouble with pregnancies, except that the first and last labours were prolonged. The physical signs of heart disease were known 30 years before; failure was recent	R.
60	58	16	6	—	Mitral stenosis and regurgitation,	Dyspnœa and œdema	3 years	Pregnancies uneventful	R.
61	59	None	3	—	Mitral stenosis, aortic regurgitation, ascites	Dyspnœa	9 months	Sent to an infirmary a wreck; in all probability died soon after	D.

62	64	16	1	—	Mitral stenosis and regurgitation,	Dyspnœa and cough	1 year	Child born soon after marriage at 23	R.
63	69	None	9	2	Mitral stenosis, pleurisy	Cough and chest pain	3 years	Pregnancies uneventful	R.
64	71	,,	4	—	Mitral stenosis	Palpitation	6 months	Pregnancies uneventful	R.
65	22	10	1	0	Mitral stenosis and regurgitation, acute rheumatism	Rheumatism	None	Child born a year before, without heart symptoms	R.
66	22	15	1	0	Mitral stenosis and regurgitation, acute rheumatism	Rheumatism	None	Child born 5 months before, without heart symptoms	R.
67	23	10	2+ 1 D.	0	Mitral stenosis and regurgitation, aortic regurgitation, acute rheumatism	Rheumatism	None	Last child 14 months ago. Has never had cardiac symptoms since pericarditis at 13 years	R.
68	25	10	2	0	Mitral stenosis and regurgitation, acute rheumatism	Rheumatism	None	Pregnancies natural	R.
69	25	20	2	0	Mitral stenosis and regurgitation, acute rheumatism	Rheumatism	None	Pregnancies natural	R.
70	27	Childhood	6	1	Mitral stenosis, acute rheumatism	Rheumatism	None	Miscarriage 1 month ago; no cardiac symptoms	R.
71	28	14	1	0	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Angina (1 year)	None	Child born without trouble	R.
72	30	7	3	—	Mitral stenosis, acute rheumatism (5th attack)	Rheumatism	None	Pregnancies natural	R.
73	30	None	0	2	Mitral stenosis, general debility for 2 years	Debility	None	Last miscarriage 8 years ago	R.
74	31	Childhood	3	1	Mitral stenosis and regurgitation, pneumonia	Pneumonia	None	Abortion 2 months ago, pneumonia followed; there were no cardiac symptoms	R.
75	35	5	5	0	Mitral stenosis and regurgitation, aortic stenosis and regurgitation, acute rheumatism (4th attack)	Rheumatism	None	Was known to have bruits at 12; has never had heart failure; last child 7 months ago	R.
76	36	15	4	—	Mitral stenosis and regurgitation, pleurisy	Pleurisy	None	Pregnancies natural	R.
77	36	9	3	—	Mitral stenosis, diabetes mellitus	Diabetes	None	Pregnancies natural	R.
78	37	Childhood	5	0	Mitral stenosis, movable kidney	Pain in loin	None	Pregnancies natural	R.
79	37	None	7	1	Mitral stenosis, hemiplegia (sudden embolism)	Hemiplegia	None	Last child 2 years ago without trouble	R.
80	37	13	1	0	Mitral stenosis, diabetes mellitus	Diabetes	None	Child born 5 years ago	R.
81	37	15	1	—	Mitral stenosis, acute rheumatism	Rheumatism	None	Pregnancy natural	R.
82	40	None	2	—	Mitral stenosis, carcinoma of liver	Malignant	None	Last pregnancy 3 years ago	Worse

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
83	40	None	3	0	Mitral stenosis and regurgitation, chronic osteoarthritis	Chronic joints	None	The chronic joint trouble (? septic synovitis) dated from a labour 2 years before; there had been no cardiac symptoms	R.
84	41	15	5	—	Mitral stenosis and regurgitation, acute rheumatism and simple stricture of œsophagus	Dysphagia	None	There had been no heart symptoms; she came in for simple stricture of œsophagus, and developed acute rheumatism in the ward	R.
85	44	14	7	—	Mitral stenosis, cerebral embolism, acute rheumatism	Hemiplegia	None	Last pregnancy was 6 years ago; there had been no cardiac symptoms; the hemiplegia was recent	R.
86	47	Childhood	4	—	Mitral stenosis, phthisis	Acute abdominal pain	None	Pregnancies uneventful; there had never been cardiac symptoms	R.
87	51	None	10	—	Mitral stenosis (old and fibrous) found p.m., admitted for perforated gastric ulcer, the mitral disease was unsuspected	Abdominal	None	Pregnancies uneventful; there had never been cardiac symptoms	D.
88	56	18	8	2	Mitral stenosis, hystero-epilepsy	Hysterical	None	Pregnancies uneventful; there had never been cardiac symptoms	R.
89	28	14	1	—	Mitral stenosis, lobar pneumonia	Pneumonia	None	Heart disease known since 14; no cardiac symptoms	R.
90	30	20	3	1	Mitral stenosis and regurgitation, ascites	Palpitation and œdema	3 months	No heart symptoms till 3 months ago; bruits known before marriage; pregnancies uneventful	R.

91	31	16	5	0	Mitral stenosis and regurgitation, enlarged liver, ascites, double pleural effusion	Dyspnœa, ascites	6 months acute, 15 years chronic	Has had dyspnœa since she was 16, when she was known to have heart disease; she married in spite of this, and has had 5 pregnancies without increase in symptoms; two of the children were short of full term, but lived; the acute symptoms definitely did not date from the last pregnancy	R.
92	33	5	3	—	Mitral stenosis and regurgitation	Dyspnœa and precordial pain	Acute	Was known to have heart disease at 13. The pregnancies caused no cardiac symptoms	R.
93	41	12	4	2	Mitral stenosis and regurgitation	Ascites and bronchitis	7 weeks	Has had dyspnœa and palpitations off and on since 13; she had no increase of symptoms during child-bearing	R.
94	41	16	4	—	Mitral stenosis and regurgitation, aortic regurgitation	Dropsy	Recent	Has had dyspnœa off and on since 16; she had no increase of symptoms during child-bearing; she was married at 19	D.
95	42	Child-hood	8	—	Mitral stenosis and regurgitation, anasarca	Dyspnœa and dropsy	8 months	The last pregnancy was 3 years ago. She was married at 22. The bruits had been known to exist for 10 years. She bore her children without cardiac symptoms, but transient hemiplegia occurred 3 days after last labour, 3 years ago	R.
96	43	None	1	—	Mitral stenosis	Dyspnœa	Acute lately	She has had dyspnœa on exertion as long as she can remember; the child was born 25 years ago without any trouble	R.
97	69	19	1	—	Mitral stenosis and regurgitation	Precordial pain and dyspnœa	Recent	Cardiac bruits known since 19	R.
98	24	8	1	0	Mitral stenosis and regurgitation, pericarditis, ascites	Pericarditis	Recent	The only pregnancy was 5 years ago, without trouble	D.
99	28	8	1	—	Mitral stenosis and regurgitation, infective endocarditis	Dyspnœa and weakness	Gradual onset for 1 year	The only pregnancy was 9 years ago, without trouble	D.
100	28	None	1	3	Mitral stenosis and regurgitation, infective endocarditis, thromboses	Œdema and dyspnœa	2 months	Married 8 years, no recent pregnancy	D.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
101	28	None	2	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation, ulcerative endocarditis	Hemiplegia, acute 3 months ago, vomiting	Diagnosed as gastric ulcer 3 months ago, no cardiac symptoms	Pregnancies uneventful	D.
102	32	10	3	—	Mitral stenosis and regurgitation, infective endocarditis	Pyrexia and joint pains	Recent	Pregnancies uneventful	D.
103	32	Childhood	5	—	Mitral stenosis and regurgitation, anasarca	Dropsy and dyspnœa	Getting worse for 1 year	Married at 16, last labour some years before admission, without difficulty	D.
104	33	None	2	0	Mitral stenosis and regurgitation, enlarged liver, etc., infective endocarditis	Dyspnœa	11 months	Last pregnancy was 3 years ago, without trouble. The mitral stenosis found p. m. was extreme	D.
105	35	,,	7	—	Mitral stenosis, pericarditis, pneumonia; the mitral stenosis was unsuspected, but was found p. m.	Pneumonia	Acute	No cardiac symptoms, pregnancies uneventful	D.
106	36	,,	4	1	Mitral stenosis, enlarged liver, etc.	Dyspnœa	A few months	Last child 8½ years ago; husband died 6 years ago	D.
107	36	,,	4	—	Mitral stenosis, aortic regurgitation, infective endocarditis	Dyspnœa and œdema	4 weeks	Pregnancies uneventful	D.
108	37	Childhood	4	—	Mitral stenosis, pleurisy, enlarged liver, œdema, etc.	Dyspnœa, dropsy	2 years, recent	Pregnancies uneventful; last some years ago	D.
109	37	17	1		Mitral stenosis and regurgitation, pericarditis, anasarca	Œdema and ascites	Gradual onset for 2 years	Last pregnancy 13 years ago	D.
110	37	16	0	1	Mitral stenosis, pleuritic effusion	Dyspnœa and œdema	On and off for 4 years acute a few months	The pregnancy was several years before	D.
111	38	14	6	—	Mitral stenosis, lobar pneumonia, empyema	Pneumonia	None before admission	Last child 9 years ago	D.
112	38	Childhood	1	—	Mitral stenosis and regurgitation, aortic regurgitation, pleuritic effusion	Dyspnœa and œdema	1 month	Married at 18. Had child without trouble	D.
113	39	,,	2	—	Mitral stenosis and regurgitation, ascites	Anasarca and orthopnœa	2 years	Had had very many attacks of rheumatism before 20. Pregnancies uneventful	D.

114	40	19	3	—	Mitral stenosis and regurgitation, enlarged liver, ascites	Dyspnœa and anasarca	A cardiac wreck for the last 4 years	The pregnancies were uneventful, and long preceded the heart failure	D.
115	40	17	10	0	Mitral stenosis, aortic stenosis, enlarged liver, infarcts, spleen and kidneys, lungs	Dyspnœa and hæmoptysis	Off and on 6 years, anasarca 1 month	Was married at 20. The last pregnancy occurred before the onset of severe hæmoptysis, though there had been slight hæmoptysis without heart failure previously	D.
116	40	15	6	1	Mitral stenosis and regurgitation, infarcts, antemortem thrombi	Dyspnœa and anasarca	Getting worse, 2 years	Pregnancies uneventful	D.
117	41	None	2	—	Mitral stenosis and regurgitation, aortic regurgitation	Precordial pain, œdema	4 months 1 month	The children were twins 21 years ago. The husband died soon after marriage	D.
118	43	14	1	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation, infective endocarditis, adherent pericardium	Rheumatic pains	Recent	The child is 19 years old	D.
119	43	None	4	Several	Mitral stenosis, tricuspid stenosis	Hemiplegia	Sudden embolism	Pregnancies uneventful	D.
120	44	,,	13	—	Mitral stenosis and regurgitation, aortic regurgitation, adherent pericardium, infarcts in kidney and spleen	Dyspnœa	Sudden onset 3 months ago	Pregnancies uneventful	D.
121	44	7	2	—	Mitral stenosis and regurgitation aortic regurgitation, tricuspid stenosis	Dyspnœa	2 years, on and off	Pregnancies uneventful, early in married life	D.
122	44	20	3	—	Mitral stenosis and regurgitation, adherent pericardium	Palpitation	3 months	Last child 8 years ago	D.
123	45	Girlhood	6	0	Mitral stenosis and regurgitation, extreme cyanosis, œdema	Dyspnœa and œdema	12 years off and on, present attack began 1 month ago	Last pregnancy preceded first cardiac symptoms by years	D.
124	46	Childhood	7	—	Mitral stenosis and regurgitation, aortic disease, infective endocarditis, enlarged liver, etc.	Œdema and dyspnœa	1 year	Husband has been dead over 6 years; pregnancies uneventful	D.
125	49	None	7	—	Mitral stenosis and regurgitation, enlarged liver, etc.	Œdema and dyspnœa	5 years	Pregnancies uneventful	D.
126	49	Childhood	8+1 D.	0	Mitral stenosis, ascites, infective endocarditis	Œdema and dyspnœa	8 months	Last child 11 years ago	D.
127	49	None	5	—	Mitral stenosis, thrombosis renal and radial arteries and aorta	Acute pains	Acute	Pregnancies uneventful	D.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
128	51	„	14	—	Mitral stenosis and regurgitation, adherent pericardium	Acute pain in chest	Acute	Pregnancies uneventful; the mitral stenosis was extreme	D.
129	52	12	2	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation, enlarged liver, ascites, pleural effusions	Dyspnœa and œdema	Recent	Pregnancies uneventful	D.
130	52	None	11	2	Mitral stenosis, tricuspid regurgitation, ascites	Anasarca	6 weeks	Pregnancies uneventful	D.
131	57	18	9	2	Mitral stenosis, œdema, etc.	Cough, œdema of legs	On and off 7 years 2 weeks	Pregnancies uneventful; on one occasion twins	D.
132	58	None	7+3D.	—	Mitral stenosis and regurgitation, ascites, etc.	Anasarca	1 year	Pregnancies uneventful	D.
133	61	„	0	4	Mitral stenosis, quite unexpected, but found p. m. There had been no bruit, kidneys healthy	Dyspnœa and weakness, œdema	2 years, recent	Pregnancies uneventful	D.
134	61	14	2	3	Mitral stenosis, aortic stenosis. tricuspid stenosis, pulmonary stenosis	Dyspnœa and weakness	2 years	Pregnancies uneventful	D.
135	71	None	11	—	Mitral stenosis found p.m., no bruit during life, kidneys sound	Œdema and bronchitis	5 months	Pregnancies uneventful	D.
136	24	„	2	1	Mitral stenosis and regurgitation, ? tricuspid stenosis	Palpitation	4 months	We are not certain of the relation, but suspect it	R.
137	26	„	4	2	Mitral stenosis and regurgitation, ascites, tec.	Ascites	1¼ years	Married 9 years. We suspect the condition was made worse by child-bearing	R.
138	33	„	6	—	Mitral stenosis, bronchitis	Bronchitis	16 years on or off	She dates her trouble from small-pox at 16. She had her first child 12 years ago, the last 9 days ago. She has had bronchitis and dyspnœa badly with each pregnancy, recovering between. The present attack has been her worst, and dates from soon after labour, 9 days ago	R.

139	37	15	12	—	Mitral stenosis and regurgitation	Dyspnœa	2 months	Eleven pregnancies were uneventful; dyspnœa came on 10 days after her twelfth labour, 2 months ago	R.
140	38	None	4	2	Mitral stenosis and regurgitation, anasarca	Œdema and dyspnœa	4 years	She had no symptoms of heart trouble until just after the last labour, an 8 months living child. 4 years ago	R.
141	39	17	4	3	Mitral stenosis and regurgitation, enlarged liver, etc.	Œdema and dyspnœa	6 years	We do not know for certain the relationship, but suspect heart trouble was made worse by pregnancies; she had been married 11 years	R.
142	39	None	6	2	Mitral stenosis, bronchitis	Cough	—	She had had bronchitis each time she was carrying; no œdema	R.
143	40	14	6	—	Mitral stenosis, pulmonary regurgitation, ascites, etc.	Œdema	11 years on and off	She dates her cardiac symptoms from soon after the birth of her second child, 11 years ago; she has borne 4 children since, each time with but slight exacerbation of her symptoms	R.
144	46	18	11	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation, ascites	Palpitation, etc.	4 years	She was quite well until 4 years ago, having borne 10 children without trouble; she dates her symptoms from shortly after the birth of her eleventh child (a living 7 months infant), 4 years ago	R.
145	47	None	3	—	Mitral stenosis and regurgitation, pleurisy	Pleurisy	Heart trouble on and off 9 years	Last child 9 years ago; the two previous gave no trouble	R.
146	26	13	2	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Dyspnœa	2 months acute	Was quite well till 3 months after birth of first child, when she had acute dyspnœa; the second pregnancy was uneventful except for persistent dyspnœa, which became acutely worse again some while after labour	R.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
147	25	None	2	0	Mitral stenosis and regurgitation. pleuritic effusion, old hemiplegia	Dyspnœa	On and off ever since a child	The last pregnancy was 2 years ago; there was no trouble with the previous child, nor indeed with the last, but the dyspnœa got gradually worse and worse after the labour; she was still alive 3 years later	R.
148	33	16	6	—	Mitral stenosis, tricuspid regurgitation	Œdema and precordial pain	8 weeks	No trouble with first 5 pregnancies, though heart disease was known from 16; 3 weeks before sixth child was born œdema of the legs began; after labour this went on to anasarca; she recovered	R.
149	21	None	1	0	Infective endocarditis, an old mitral stenosis	Hæmoptysis, and splenic pain	6 weeks	No trouble with pregnancy or labour; is now 5 months pregnant; went out, still pregnant, against advice	Worse
150	23	12	4	—	Mitral stenosis and regurgitation, ascites, etc.	Dyspnœa	3 years, acute 3 weeks	The 4 children were born without trouble, but 3 weeks ago, 2 months after last labour, acute dyspnœa set in	R.
151	24	Girlhood	1	—	Mitral stenosis, bronchitis	Cough, no œdema	Recent	Was pregnant 5½ months on admission; she got much better and went out; relapsed, came in again, recovered, went out again, and went to term without further trouble	R.
152	24	None	0	0	Mitral stenosis	Dyspnœa and hæmoptysis	3 months	Was pregnant 5 months on first admission; got better on treatment, went out, relapsed, came in again, got better, went out again still pregnant	R.

153	24	16	1 d.	—	Mitral stenosis, aortic regurgitation	Dyspnœa (not bad)	Years	Married 12 months. Had been in and out of hospital seven times for heart disease before marriage. She was in bed in hospital 203 days; was then delivered of a dead 8-months fœtus, and went out 14 days after labour pretty well	R.
154	27	17	0	1	Mitral stenosis, ascites, tapped	Ascites	1½ years	She has been married 2 years. Ascites developed during first pregnancy and caused miscarriage. She has had œdema and ascites on and off ever since	R.
155	27	None	4 + 1 d.	—	Mitral stenosis	Dyspnœa, hæmoptysis	14 months, 6 months	She bore 3 children without trouble. The fourth was 4 years ago; 5 months before this labour she had a cerebral embolism with hemiplegia. She got better of this, and had no heart trouble till 14 months ago, when dyspnœa began; 7½ months she became pregnant again, and 1½ months later hæmoptysis started. Cough increased during pregnancy, but she went almost to term, and had a living child weighing 6 lb. 8 oz., natural delivery. She went out fairly well	R.
156	28	16	4	—	Mitral stenosis and regurgitation, ascites, pleuritic effusion	Ascites	5 months	The first 3 labours were natural. Soon after the fourth child was born cardiac trouble began	R.
157	28	None	1 + 1 d.	—	Mitral stenosis and regurgitation, ascites	Dyspnœa and ascites	6 months	She had no trouble with first pregnancy, 6 years ago. Soon after the second, 6 months ago, ascites began and increased	R.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
158	29	None	4	2	Mitral stenosis, aortic regurgitation, pleuritic effusion	Hæmoptysis, severe dyspnœa	3 years on and off, 1 month	The third full-term child was 3 years ago; she dates hæmoptysis from then. After that she had two miscarriages; a month ago she was delivered of her fourth full-term child, living, and has been in bed with severe dyspnœa since	R.
159	30	Childhood	2	0	Mitral stenosis and regurgitation, tricuspid regurgitation, etc.	Severe dyspnœa	11 months	There was no trouble with the first child; the second was born 11 months ago, and following labour the dyspnœa set in	R.
160	31	None	3	0	Mitral stenosis and regurgitation, œdema	Bronchitis, œdema	Some years on and off. 20 months	Was quite well till after first labour; bronchitis then set in, and recurred with each of the two pregnancies; the last labour was 20 months ago; œdema set in after this last labour. Infective endocarditis was suspected on last admission, on account of pyrexia; she went home worse	Worse
161	32	Girlhood	5	0	Mitral regurgitation	Hæmoptysis	Recent	She was in hospital at 18 for palpitation and dyspnœa. She married subsequently, and had 4 children without trouble. When 6 months pregnant of fifth child she had sudden hæmoptysis, lasting 4 days. There was no other cardiac trouble; she only lay up 4 days; she went to term naturally	R.

162	33	None	4	—	Mitral stenosis, bronchitis	Dyspnœa and bronchitis	8 months, worse 1 week	Three pregnancies gave no trouble; early in the fourth dyspnœa and hæmoptysis set in; she went to term, and the child was born alive; the dyspnœa got worse after labour; she came to hospital for relief and recovered with rest in bed	R.
163	33	Child-hood	7	1	Mitral stenosis	Dyspnœa	Many years	The first four children caused no heart symptoms; with fifth and sixth there was dyspnœa. She is now pregnant 7 months, having had hæmoptysis for 7 months; œdema set in at 7 months, and got worse to term. The child was born living naturally; there was a bad attack of dyspnœa on fourth day after labour; the mother responded to treatment, and went out moderately well	R.
164	34	20	7	1	Mitral stenosis and regurgitation, infarcts in spleen and lungs	Œdema and ascites	4 months	She dates her heart failure directly to her miscarriage 4 months ago, when 6 months pregnant. There was no trouble with any of previous 7 children	D.
165	34	Child-hood	2	0	Mitral stenosis, pleurisy	Pleurisy	None	She came in with a week's history of pleuritic pain when pregnant nearly to term. A pleuritic effusion was found. Labour at term was natural	R.
166	36	None	2	0	Mitral stenosis, tricuspid stenosis	Dyspnœa	4 months	No trouble with pregnancy till fourth month, when acute dyspnœa set in. She had several attacks of dyspnœa, but went to term, and was delivered of living twins (boys) naturally. Two weeks after labour there was another very acute attack of dyspnœa; the patient rallied rapidly, and went out apparently well	R.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
167	37	None	5	1	Mitral stenosis and regurgitation, ascites, etc.	Orthopnœa and œdema	9 weeks	She was quite well during five former pregnancies, but had a miscarriage 9 weeks ago, since when she has not been well	R.
168	37	None	12	—	Mitral stenosis and regurgitation	Acute rheumatism	No real heart failure	She had no trouble at all with the first 12 children; when 4 months pregnant with the 13th she got very bad rheumatic fever, and was found to have signs of old mitral disease. She recovered and went out still pregnant	R.
169	38	20	6	—	Mitral stenosis and regurgitation	Œdema and dyspnœa	Many years on and off	She got dyspnœic during her first pregnancy, and has been bad with each subsequently. On two occasions labour was induced at the 8th month for heart failure, on one of which occasions p. p. h. was almost fatal. After her fourth child she was discharged "a wreck," but recovered at home, and bore two more children. The last of these was born without induction; it was a transverse presentation; version was performed; the mother and child both did well	R.
170	38	12	10	—	Mitral stenosis and regurgitation	Dyspnœa	14 months	There was no trouble with first nine children; after the birth of the tenth, 14 months ago, dyspnœa set in, and has been getting worse and worse since	R.

171	41	16	5 + 1 D.	2	Aortic disease and mitral stenosis	Dyspnœa	Recent	No trouble with former labours. When pregnant for eighth time, and near to term, dyspnœa began, followed by easy labour and recovery. The child was dead	R.
172	43	18	13	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Œdema	5 months	12 pregnancies were uneventful; with the 13th cardiac symptoms began; œdema set in 2 months after labour, and grew worse. She recovered with rest in bed	R.
173	41	None	7	—	Mitral stenosis and regurgitation.	Anginal pain	14 years	3 pregnancies were uneventful; after the fourth labour anginal attacks began. Notwithstanding these, she bore three more living children, the last six years ago. She is a chronic invalid	Very ill
174	25	None	1	—	Mitral stenosis, thromboses, anasarca	Dyspnœa	14 days	The child was born living 14 days ago; there were no symptoms till after labour	D.
175	38	„	8	—	Mitral stenosis and regurgitation, calcareous vegetations	Dyspnœa	11 weeks	Was quite well until after last confinement, 11 weeks ago; the first 7 pregnancies were uneventful	D.
176	31	„	8	—	Mitral stenosis, pleuritic effusion	Anasarca, precordial pain, dyspnœa	7 weeks, many years on and off	She had cardiac symptoms shortly after second pregnancy, and was short of breath through all the subsequent ones; the first was natural, the last was 7 weeks ago	D.
177	31	„	5	1	Mitral stenosis and regurgitation, aortic stenosis, infarcts in lungs	Dyspnœa and œdema	1 year	She came in pregnant and got better under treatment; she went out and went to term naturally; she came in again a few weeks afterwards. She dated her heart symptoms to the miscarriage 1 year ago	D.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
178	30	17	1	0	Mitral stenosis, tricuspid stenosis, aortic stenosis, infarcts in lungs, gastric ulcer	Anasarca, dyspnœa, and hæmatemesis	Some months	The cardiac symptoms came on early in the only pregnancy, but she rested and went to term; the child was small, living; she collapsed 10 days after labour, a few days before admission; she got worse and worse, and died	D.
179	25	None	2	2	Mitral stenosis, infective endocarditis, various infarcts	Œdema and weakness, and acute hemiplegia	Recent, some years	The first labour at term was natural; then followed two miscarriages, and there were cardiac symptoms with each; the last labour at term was 10 months ago, without much trouble, but the patient has never been well since; the progress was downhill continuously	D.
180	23	„	3	1	Mitral stenosis, infective endocarditis, various infarcts	Chorea	Recent	There had been no previous chorea; the three children were born without trouble. When pregnant fourth time, she developed chorea at sixth month and aborted 21 days afterwards; she went rapidly downhill and died 23 days after the abortion	D.
181	31	12	5	—	Mitral stenosis and regurgitation, adherent pericardium, ascites, etc.	Cough, œdema	9 years, 2 years	The first 4 children were born without trouble; the fifth was born alive at term 3 months ago naturally; œdema of legs and ascites came on one week after labour; the patient went rapidly downhill	D.

182	20	14	1	0	Mitral stenosis and regurgitation, infective endocarditis, pericarditis	Œdema	2½ years	Symptoms of heart failure came on early during the only pregnancy, 2½ years ago; the cardiac symptoms were so bad that labour was induced at the eighth month; the child lived. The mother recovered a little, but was a chronic invalid, and finally developed malignant endocarditis	D.
183	28	None	4	0	Mitral stenosis, tricuspid regurgitation	Dyspnœa	Some months	She "had never been ill in her life" until, when 4 months pregnant of the fourth child, symptoms of dyspnœa and cough came on; after rest and digitalis in hospital she got better and went home. She returned at term, and had a living child easily; the mother did well at first, but a few days after getting up she developed further heart symptoms, and rapidly went downhill and died	D.
184	24	16	1	—	Mitral stenosis and regurgitation. bronchitis	Dyspnœa and œdema	18 months	Heart failure began during the pregnancy, but acute symptoms did not arise until a living child had been born at term. Since then she had been in and out of hospital 5 times in a year, never really recovering compensation	D.
185	40	None	4	4	Mitral stenosis, infarcts in kidneys	Dyspnœa	5 months	She had always been well, except that 10 years ago she was in hospital for albuminuria during pregnancy. Four labours and 3 miscarriages were without cardiac symptoms; the latter date from a miscarriage at the 3rd month, 5 months ago	D.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
186	28	Childhood	1	—	Mitral stenosis, tricuspid stenosis, aortic regurgitation	General failure	2 years	She was quite well till the child was born, 2 years ago; heart failure set in soon after labour, and she has never been well since	D.
187	28	None	1	—	Mitral stenosis, hemiplegia, infarcts, tricuspid vegetations	Palpitation and hæmoptysis	5 years	Palpitation and hæmoptysis have recurred during the last five years. The only child was born living at the 7th month, 8 months ago, and the cardiac symptoms became much worse. She has gone downhill ever since	D.
188	32	17	2	—	Mitral stenosis and regurgitation, hemiplegia	Œdema	3 years on and off	The first child was born normally. The symptoms date from soon after the birth of the second child, 3 years ago	D.
189	38	6	1	—	Mitral stenosis, infective endocarditis, pleuritic effusion, various infarcts	Dyspnœa and hæmoptysis	7 months	The child was born naturally 9 months ago. Two months later dyspnœa and hæmoptysis set in; the heart symptoms went from bad to worse	D.
190	41	8	6 + 1 D.	1	Mitral stenosis and regurgitation, infective endocarditis	Œdema, dyspnœa, acute	9 months, 2 weeks	There was no trouble till the last child was born, 2 years ago. Soon after she had hemiplegia. No other cardiac symptoms followed until 9 months ago, when œdema appeared; she became acutely dyspnœic 2 weeks ago and died in a few weeks	D.

191	43	8	2	—	Mitral stenosis and regurgitation, vegetations, œdema, hæmoptysis	Dyspnœa, œdema	$1\frac{3}{4}$ years, 1 month	The first child brought no heart trouble. Three months after the birth of the second, $1\frac{3}{4}$ years ago, the patient became dyspnœic. She was able to do her work until 1 month ago, when œdema came on, and she died soon after admission. It is doubtful if this can really be attributed to the pregnancy	D.
192	48	Girl-hood	8	4	Mitral stenosis and regurgitation, general heart failure	Dyspnœa	10 years	The patient directly dates symptoms to a labour 10 years ago. She has since been pregnant 3 times. The eldest child is 25, the youngest 6. She has never been well since the last was born, though she has done her work on and off till recently	D.
193	25	18	—	—	Mitral stenosis and regurgitation	Rheumatism	None	Married 3 years	R.
194	26	None	—	—	Mitral stenosis and regurgitation	Dyspnœa and precordial pain	2 years	Married recently. Heart troubles started before marriage	Worse
195	26	Girl-hood	—	—	Mitral stenosis, bronchitis	Œdema and cough	5 weeks	—	D.
196	29	None	—	—	Mitral stenosis, tricuspid regurgitation	Dyspnœa and weakness	6 years	Married 9 years	D.
197	31	16	—	—	Mitral stenosis and regurgitation, bronchitis	Cough and precordial pain	2 months	Married 11 years	R.
198	34	None	—	—	Mitral stenosis and regurgitation, pulmonary regurgitation, ascites, etc.	Œdema and ascites	2 months	Married 8 years, and has been out of health on and off ever since	R.
199	34	„	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Dyspnœa and œdema	4 months	—	R.
200	35	16	—	—	Mitral stenosis, general failure	Dyspnœa	18 months	Has been in Guy's Hospital more than a dozen times. She recovers quickly, but soon relapses. She is a widow	R.
201	36	7	—	—	Mitral stenosis, anasarca	Dyspnœa and ascites	1 year	Had cerebral embolism 9 years ago. Married 18 years	D.
202	36	None	—	—	Mitral stenosis and regurgitation, tricuspid regurgitation	Orthopnœa and œdema	17 years	Married 8 years. A chronic hospital inmate	R.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
203	39	,,	—	—	Mitral stenosis, epithelioma of œsophagus	Dysphagia	None	Married 9 years. Mitral stenosis, unsuspected, found p. m.	D.
204	41	4	—	—	Mitral stenosis and regurgitation, double aortic disease	Dyspnœa	Months	—	D.
205	55	12	—	—	Mitral stenosis, hæmatemesis	Dyspnœa and cyanosis	28 years on and off	—	D.
206	20	6	—	—	Mitral stenosis	Dyspnœa, hæmoptysis	2 years	—	R.
207	20	None	—	—	Mitral stenosis, aortic stenosis	Dyspnœa and palpitations	2 months	—	R.
208	20	,,	—	—	Mitral stenosis	Dyspnœa	3 years	—	R.
209	21	Child-hood	—	—	Mitral stenosis, acute rheumatism	Precordial pain	None	—	R.
210	21	19	—	—	Mitral stenosis and regurgitation, acute rheumatism	Dyspnœa	2 years on and off	Hemiplegia due to cerebral embolism occurred just before admission	R.
211	21	12	—	—	Mitral stenosis and regurgitation	Dyspnœa	Acute	—	R.
212	21	Girl-hood	—	—	Mitral stenosis and regurgitation, aortic stenosis, acute rheumatism	Precordial pain	3 months	—	R.
213	21	12	—	—	Mitral stenosis and bronchitis	Cough and dyspnœa	7 years	—	R.
214	21	None	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Precordial pain	Acute	—	R.
215	21	10	—	—	Mitral stenosis and regurgitation, bronchitis	Cough and pain in chest	2 weeks	—	R.
216	21	10	—	—	Mitral stenosis and regurgitation, anasarca	Dyspnœa and œdema	Recent	—	R.
217	21	7	—	—	Mitral stenosis and regurgitation	Pain in side, œdema	Recent	—	R.
218	22	14	—	—	Mitral stenosis and regurgitation, paracentesis abdominis	Precordial pain, ascites	1 year	—	R.
219	22	10	—	—	Mitral stenosis and regurgitation	Dyspnœa	Recent	—	R.
220	22	10	—	—	Mitral stenosis and regurgitation, large liver, etc.	Œdema	1 month	—	R.
221	22	None	—	—	Mitral stenosis and regurgitation, bronchitis	Dyspnœa and œdema	6 months	Was often admitted afterwards A chronic invalid	R.

222	22	11	—	—	Mitral stenosis and regurgitation	Acute rheumatism	None	—	R.
223	23	11	—	—	Mitral stenosis and regurgitation, infective endocarditis	Hæmaturia	Recent	Went home to die	Worse
224	23	12	—	—	Mitral stenosis, epilepsy	Fits	None	—	R.
225	23	None	—	—	Mitral stenosis and regurgitation, rheumatic nodules	Weakness	Months	—	R.
226	23	Childhood	—	—	Mitral stenosis, hæmatemesis	Hæmatemesis	None	—	R.
227	23	14	—	—	Mitral stenosis and regurgitation	Œdema	5 months	—	R.
228	23	14	—	—	Mitral stenosis and regurgitation	Dyspnœa	Recent	—	R.
229	23	Childhood	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Dyspnœa	4 years	—	R.
230	23	16	—	—	Mitral stenosis and regurgitation, pericarditis	Dyspnœa	3 months	—	R.
231	23	None	—	—	Mitral stenosis and regurgitation, exophthalmic goitre, Raynaud's disease	Nervousness	None	—	R.
232	23	16	—	—	Mitral stenosis and regurgitation, bronchitis	Cough and dyspnœa	4 months	—	R.
233	24	8	—	—	Mitral stenosis and regurgitation, gastritis	Gastric pain and vomiting	None	—	R.
234	24	7	—	—	Mitral stenosis and regurgitation, acute rheumatism (3rd attack)	Rheumatism	None	—	R.
235	24	19	—	—	Mitral stenosis and regurgitation, gastritis	Gastric pain	None	—	R.
236	24	None	—	—	Mitral stenosis, ganglion on wrist	Ganglion	None	—	R.
237	24	9	—	—	Mitral stenosis and regurgitation, nutmeg liver	Dyspnœa and œdema	Years	—	R.
238	24	16	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Dyspnœa	Years	—	R.
239	25	9	—	—	Mitral stenosis and regurgitation, ascites	Dyspnœa	2 months	—	R.
240	25	None	—	—	Mitral stenosis and regurgitation	Œdema	1 year	Palpitation for 7 years	R.
241	25	16	—	—	Mitral stenosis and regurgitation	Precordial pain and œdema	Recent	—	R.
242	25	None	—	—	Mitral stenosis and regurgitation	Palpitation	1 year	—	R.
243	25	,,	—	—	Mitral stenosis and regurgitation	Dyspnœa and œdema	4 years	—	R.
244	26	20	—	—	Mitral stenosis and regurgitation, pleuritic effusion	Dyspnœa and œdema	1 year	There was slight cerebral embolism 1 year ago	R.
245	26	None	—	—	Mitral stenosis and regurgitation, pericarditis	Dyspnœa and œdema	10 months	—	R.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of Children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result
246	27	7	—	—	Mitral stenosis and regurgitation	Œdema	2 weeks	—	R.
247	27	12	—	—	Mitral stenosis and regurgitation, aortic regurgitation	Bad dyspnœa and cough	Years	Has been in and out of hospital nearly a dozen times	A wreck
248	27	7	—	—	Mitral stenosis and regurgitation, aortic stenosis	Dyspnœa and palpitation	7 months	—	R.
249	28	10	—	—	Mitral stenosis and regurgitation	Œdema	2 years	—	R.
250	28	16	—	—	Mitral stenosis, chronic osteo-arthritis	Deformed joints	None	—	R.
251	28	12	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Pleuritic effusion	Acute	—	R.
252	28	Child-hood	—	—	Mitral stenosis, tricuspid regur-gitation	Dyspnœa and œdema	1 year	—	R.
253	28	None	—	—	Mitral stenosis and regurgitation, cerebral embolism	Hemiplegia	None	—	R.
254	29	11	—	—	Mitral stenosis and regurgitation, aortic regurgitation, enlarged liver	Orthopnœa	4 years	—	R.
255	30	10	—	—	Mitral stenosis and regurgitation, aortic regurgitation	Hæmoptysis	Recent	—	R.
256	30	6	—	—	Mitral stenosis	Precordial pain	4 years	—	R.
257	31	18	—	—	Mitral stenosis	Hæmoptysis	None	—	R.
258	31	14	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Dyspnœa	1 year	—	R.
259	32	None	—	—	Mitral stenosis and regurgitation, enlarged liver, etc., ascites	Orthopnœa	6 years	—	Worse
260	32	12	—	—	Mitral stenosis and regurgitation, pleurisy, typhoid fever	Enteric	None	—	R.
261	32	None	—	—	Mitral stenosis	Dyspnœa, hæmoptysis	6 months	—	R.
262	33	„	—	—	Mitral stenosis and regurgitation, bronchitis	Dyspnœa	1 year	—	R.
263	33	16	—	—	Mitral stenosis and regurgitation	Pleurisy	None	—	R.
264	34	7	—	—	Mitral stenosis and regurgitation	Dyspnœa	Years	Known to have had heart disease at 15	R.

265	34	None	—	—	Mitral stenosis, transverse myelitis	Paraplegia	None	—	R.
266	35	„	—	—	Mitral stenosis, carcinoma of breast	Dyspnœa	11 years	—	R.
267	35	5	—	—	Mitral stenosis, appendicitis	Appendicitis	None	—	R.
268	35	16	—	—	Mitral stenosis and regurgitation, tricuspid regurgitation	Dyspnœa	9 years	Known to have had heart disease at 14	R.
269	35	Childhood	—	—	Mitral stenosis and regurgitation, aortic regurgitation	Dyspnœa	3 years	—	R.
270	35	None	—	—	Mitral stenosis	Precordial pain	8 months	—	R.
271	36	„	—	—	Mitral stenosis	Cough and weakness	1 year	—	R.
272	37	„	—	—	Mitral stenosis, acute rheumatism, mania	Insanity	None	—	R.
273	37	16	—	—	Mitral stenosis, cerebral embolism	Hemiplegia	6 years	—	R.
274	38	Girlhood	—	—	Mitral stenosis, gastric ulcer	Hæmatemesis	None	—	R.
275	39	20	—	—	Mitral stenosis, mad with delusions	Hæmoptysis	11 years, on and off	—	R.
276	40	None	—	—	Mitral stenosis, aortic regurgitation	Dyspnœa	6 weeks	—	R.
277	40	19	—	—	Mitral stenosis, tricuspid regurgitation, bronchitis	Dyspnœa and cough	3 years	—	R.
278	40	None	—	—	Mitral stenosis, pelvic tumour, no operation	Dyspnœa	Many years	—	R.
279	43	„	—	—	Mitral stenosis and regurgitation, aortic stenosis	Dyspnœa	Years	—	R.
280	45	Childhood	—	—	Mitral stenosis and regurgitation	Dyspnœa and œdema	4 months	—	R.
281	47	20	—	—	Mitral stenosis and regurgitation, enlarged liver, etc.	Dyspnœa and pain	Years	—	R.
282	48	None	—	—	Mitral stenosis and regurgitation, enlarged heart, bronchitis	Dyspnœa and cough	1 year	—	R.
283	60	„	—	—	Mitral stenosis and regurgitation	Dyspnœa and pain	1 month	—	R.
284	21	None	—	—	Mitral stenosis and regurgitation, aortic regurgitation	Dyspnœa	1½ years	—	D.
285	23	9	—	—	Mitral stenosis, pericarditis	Dyspnœa	1 year	—	D.
286	23	None	—	—	Mitral stenosis, infective endocarditis, infarcts	Malaise	Recent	—	D.

Case Number.	Age.	Age at which rheumatism or chorea.	Number of children.	Number of miscarriages.	Main diagnosis.	Symptoms for which admitted.	Duration of cardiac failure.	Details.	Result.
287	23	16	—	—	Mitral stenosis and regurgitation, acute endocarditis	Dyspnœa	7 years	—	D.
288	23	None	—	—	Mitral stenosis, pericarditis, infective endocarditis	Dyspnœa and pain	4 months	—	D.
289	24	12	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation	Angina pectoris	6 years	—	D.
290	26	None	—	—	Mitral stenosis (old), infective endocarditis	Dyspnœa	2 years	—	D.
291	26	„	—	—	Mitral stenosis, pericarditis, pleurisy, exophthalmic goitre	Joint pains	None	—	D.
292	28	Childhood	—	—	Mitral stenosis, tricuspid stenosis, dropsy	Dropsy	3 years	—	D.
293	29	15	—	—	Mitral stenosis, pericarditis, pleuritic effusion, infarcts	Dyspnœa	Recent	—	D.
294	33	None	—	—	Mitral stenosis, infective endocarditis, infarcts	Sudden hemiplegia	Recent	—	D.
295	38	„	—	—	Mitral stenosis and regurgitation, enlarged liver, infarcts	Dyspnœa	2 years	—	D.
296	40	Childhood	—	—	Mitral stenosis and regurgitation, adherent pericardium	Dyspnœa	Recent	—	D.
297	42	None	—	—	Mitral stenosis and regurgitation, tricuspid stenosis, anasarca	Dropsy	Recent	—	D.
298	44	„	—	—	Mitral stenosis, enl'rg'd liver, ascites, pericarditis, pleuritic effus'n	Dropsy	Recent	—	D.
299	44	12	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation, adherent pericardium	Dyspnœa	Years	—	D.
300	41	19	—	—	Mitral stenosis and regurgitation, aortic stenosis and regurgitation, infective endocarditis	Dyspnœa	4 months	—	D.

Fifty Cases of Labour Complicated by Organic Disease of the Heart, from the Records of Queen Charlotte's Hospital, with a Note on the Blood Pressure during Pregnancy.

BY

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AND

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AN important paper recently brought before the Royal Medical and Chirurgical Society by Dr. French and Mr. Hicks went far to throw doubt upon the generally accepted teaching that pregnancy and labour entail grave danger to life in women suffering from mitral stenosis. At the same meeting, Dr. Griffith made some remarks founded upon his own experience and upon a number of cases collected for the purpose from the records of Queen Charlotte's Hospital. The Editor of this JOURNAL suggested that these cases should be cast into the form of a paper which would present the subject more from the obstetrical point of view. We have therefore added considerably to the original number of cases. It is necessary to state that the majority of the patients remained in the hospital for fourteen days only, and we are not in a position to state whether any deaths occurred after they had left the hospital. Up to the present time no routine examination of the heart has been carried out at Queen Charlotte's Hospital, and therefore only those cases in which attention was in some way attracted to the cardiac condition have been recorded; it is probable that others have escaped notice. We have looked through the records of 11,319 patients in order to collect the fifty cases, and no case has been accepted unless the physical signs of organic heart disease were present.

Maternal Mortality. Four cases, or eight per cent. terminated fatally while under observation in the Hospital. A short account of each of these fatal cases is given.

CASE I. Primigravida, aged 22. Three years before admission the patient was in a general hospital with swollen wrists and ankles.

Eight weeks before admission to Queen Charlotte's she had "influenza" which left her heart weaker than formerly. She had had an oscillating high temperature for some weeks before admission and was delirious during the last week. On admission the heart showed signs of failing compensation, the cardiac area being enlarged and the pulse very irregular. The murmurs and other signs of double mitral disease were present. Labour came on spontaneously at the end of the eighth month and was assisted by forceps. After delivery the patient improved considerably, but the fever did not leave her, and she died on the 28th day after delivery. *P.M.:* Ulcerative endocarditis and infarcts in the kidneys and spleen were found.

Case II. Primigravida, aged 24, suffering from mitral stenosis and regurgitation, was admitted in a critical condition, compensation having entirely broken down. Chloroform was given and the os manually dilated; forceps extraction failed and the infant (which was dead) was delivered by craniotomy. Fifteen fluid ounces of blood were lost. The patient was severely collapsed after delivery and died in three quarters of an hour. Labour took place three weeks before term.

Case III. A 10-gravida, aged 43, was admitted suffering from mitral regurgitation of rheumatic origin. Compensation had broken down, œdema and bronchitis being prominent symptoms; all previous labours normal. Labour in this case was very short, the whole process being completed without assistance or abnormal loss of blood in 25 minutes. Ten minutes after delivery, the patient became unconscious and cyanotic, and death ensued. The infant did well.

Case IV. A 6-gravida, aged 40, all previous labours normal, suffered from mitral regurgitation. At the 28th week labour pains came on unexpectedly, and as the patient was getting into a cab to come to hospital, the baby was suddenly born, labour in this case also being precipitate. She suffered from considerable distress after delivery from which she recovered moderately well, but increasing cardiac incompetence brought about a fatal termination on the 19th day of the puerperium.

Infantile Mortality. In the series of primiparæ five infants were born dead. One was macerated, two were perforated, and the other two were premature. In the second series of multiparæ five infants died, none of which was viable.

Average Parity of Multiparæ. Thirty-one multiparæ had had 159 children amongst them, the average parity being therefore about five apiece. Twenty of the patients had had four or more children.

Miscarriage and Premature Labour. In six instances only did we find any statement of previous miscarriages. Premature labour took place in five primiparæ and eleven multiparæ, being brought on in six instances by the introduction of bougies. In ten of these sixteen cases the pregnancy had lasted over thirty-two weeks, and in six, under thirty-two weeks.

Type of Heart Disease. There was a definite history of rheumatic fever or chorea in 32 instances. In the other cases no statement bearing on the origin of the cardiac lesion was made. The following table shows the varieties of heart disease:—

	Mitral stenosis	Mitral stenosis and regurgitation	Mitral regurgitation	Mitral and aortic Disease
Primiparæ ...	1	8	8	2
Multiparæ ...	5	12	13	1

Condition of Compensation before Labour. Taking into consideration the ordinary signs of failing compensation we have roughly classified the cases as follows:—

	Good.	Poor.	Bad.
Primiparæ	14	2	3
Multiparæ	21	6	4

	Albuminuria (Catheter Specimens).			
	No Note.	Nil.	Trace.	Large Amount.
Primiparæ ...	4	7	3	5
Multiparæ ...	4	20	4	3

Delivery. Labour took place without assistance in eleven primiparæ and in twenty-three multiparæ, or in 68 per cent. of the total number of cases. Chloroform was given and the child delivered by forceps in 8 primiparæ and 6 multiparæ. Labour was induced by bougies in three primigravidæ and three multigravidæ with good results. In two cases the child was perforated and in one case the cervix was dilated with a bag and the child delivered by version. In two cases the pelvis was contracted. In two, both of which did well in all respects, twins were born. In two more the placenta was morbidly adherent and was manually removed.

Duration of Labour in Hours and Minutes:—

	1st Stage.	2nd Stage.	3rd Stage.	Total.
Primiparæ	16·21	0·48	0·16	17·25
Multiparæ	8·45	0·20	0·14	9·19

Bleeding. The amount of blood lost after the birth of the child was rather above the normal, averaging eleven fluid ounces in the primiparæ and twelve in the multiparæ. Two of the primiparæ lost 25 and 40 fluid ounces respectively, while one of the multiparæ lost 60 fluid ounces and five lost 20 fluid ounces each.

Condition immediately after Delivery. (1) Primiparæ. In two cases the condition of the patient after delivery was serious, one, case 6, died, and the other, case 4 (twins), was distressed for some hours after delivery but made a good recovery subsequently. In the remaining 17 cases the condition after delivery was satisfactory. (2) Multiparæ. In 21 instances the patients passed through labour without any sign of distress. In five cases there was a slight and transient condition of distress, and in five more (two of whom died) there was severe distress. Cyanosis and dyspnœa were the most common symptoms.

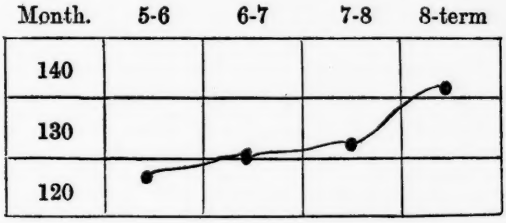
Puerperium. Two deaths took place late in the puerperium, one a primipara dying of ulcerative endocarditis on the 28th day of the puerperium, and the other a multipara, of gradually increasing cardiac incompetence on the 19th day. Six of the patients suffered from slight dyspnœa during the puerperium, and three had symptoms of sapræmia.

Note on the Blood Pressure during Pregnancy.

By C. N. Longridge.

Having been engaged for some little time upon an investigation into the variations of blood pressure in pregnancy, labour and the puerperium, I thought that, although this investigation is not yet complete, it might not be out of place to add to this paper a short note on the subject. Sufficient data have been obtained to allow of definite statements upon the variations of blood pressure during pregnancy being made. The observations were made in the out-patient room of Queen Charlotte's Hospital upon 142 primigravidæ who came up to be examined. As each patient only comes up once for examination, continuous observations on the same patient could

not be carried out. The readings were taken by means of Martin's modification of the Riva Rocci apparatus, the bag being fixed on the right arm and the patient being in the recumbent position in all cases. The patient's apprehensions were soothed as far as possible before the readings were taken. The results, which are set forth in the accompanying table, show that a slight but distinct rise of blood pressure takes place in the latter months of pregnancy.



(The normal blood pressure is generally taken as equal to 120 mm. of mercury.)

This rise appears to me to have a definite bearing on the question of hypertrophy of the heart during pregnancy. For whatever may be the cause of the rise of blood pressure, it seems that the heart under the circumstances could not act otherwise than in accordance with ordinary physiological laws, and meet the increased work entailed by the raised blood pressure by hypertrophy of its muscular tissue. The fact that so many of our cases reached the end of pregnancy and passed through labour without any failure of compensation appears to favour the view that actual hypertrophy of the heart does take place.

SERIES I.—Primiparæ.

No.		Parse.	Age.	Type.	Term.	Compensation.	Alb.	Spontaneous.	Forceps.	Chloroform.	Other measures.	Distress after.	Result. M.	Result. C.	Remarks.
1	C. K.	...	18	Aortic and Mitral.	Full	Good	No note	–	+	+	Perforation	—	g.	D.	Contracted pelvis, C.V. 3¾. Prolapse of cord.
2	G. G.	...	22	Mitral Stenosis and Regurgitation.	36/52	Bad	Nil	–	+	+	—	—	D.	g.	Mother died of endocarditis 28th day.
3	M. D.	...	32	Mitral Regurgitation.	35/52	Good	¾	–	+	+	Induction	—	g.	D.	Induction for attacks of dyspnœa. Infant,
4	R. E.	...	30	Mitral Stenosis.	Full	Poor	Solid	–	+	+	—	Mod.	g.	g.	Twins. [macerated knot in cord.
5	A. C.	...	25	Mitral Stenosis and Regurgitation.	30/52	Bad	Trace	–	+	+	—	—	g.	D.	Infant not viable.
6	W. B.	...	24	Mitral Stenosis and Regurgitation.	37/52	Bad	No note	–	+	+	Perforation	Bad	D.	D.	Condition critical on admission. Macerated
7	E. C.	...	32	Mitral Regurgitation.	Full	Good	½	+	–	–	—	—	g.	g.	[perforation.
8	M. P.	...	31	Mitral Stenosis and Regurgitation.	36/52	Good	¼	+	–	–	Induction	—	g.	D.	Induced for nephritis.
9	A. G.	...	19	Mitral Regurgitation.	Full	Good	Nil	–	+	+	—	—	g.	g.	
10	E. C.	...	24	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	
11	A. R.	...	17	Aortic and Mitral Regurgitation.	Full	Poor	Trace	+	–	–	—	—	g.	g.	Slight faintness occasionally during puer-
12	A. C.	...	25	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	[perium.
13	K. C.	...	20	Mitral Stenosis and Regurgitation.	Full	Good	Trace	–	+	+	Induction	—	g.	g.	
14	J. H.	...	23	Mitral Regurgitation.	Full	Good	No note	+	–	–	—	—	g.	g.	
15	A. S.	...	15	Mitral Regurgitation.	Full	Good	No note	+	–	–	—	—	g.	g.	
16	E. H.	...	25	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	
17	A. W.	...	23	Mitral Stenosis and Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	Bronchitis in puerperium.
18	F. E.	...	27	Mitral Stenosis and Regurgitation.	Full	Good	Solid	+	–	–	—	—	g.	g.	Sapræmia.
19	H.W,	...	18	Mitral Stenosis and Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	

SERIES II.—Multiparæ.

1	A. M.	4	33	Mitral Regurgitation.	38/52	Poor	Nil	–	–	+	Induction Breech	—	g.	g.	Induction by bougies.
2	K. B.	7	22	Mitral Stenosis and Regurgitation.	Full	Good	Nil	–	+	+	—	—	g.	g.	
3	M. M.	10	43	Mitral Regurgitation.	Full	Poor	½	+	–	–	—	Bad	D.	g.	Rapid labour. Sudden unconsciousness. Cyan-
4	E. S.	8	37	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	[osis and death soon after.
5	E. W.	2	28	Mitral Stenosis and Regurgitation.	Full	Good	No note	+	–	–	—	—	g.	g.	
6	A. L.	2	30	Mitral Stenosis and Regurgitation.	36/52	Fair	Nil	–	+	+	Induction	Bad	g.	g.	Con. pelv. C.V. = 3¼. Cyanosis and distress at
7	R. W.	3	29	Mitral Stenosis and Regurgitation.	24/52	Good	Nil	+	–	–	—	Slight	g.	D.	Adher. placen. Inf. not viable. [lab. Ad. pla.
8	A. P.	3	33	Mitral Stenosis and Regurgitation.	Full	Bad	Trace	+	–	–	—	Slight	g.	g.	P.P.H., improved after bleeding.
9	K. T.	4	23	Mitral Stenosis and Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	
10	A. C.	2	26	Mitral Stenosis and Regurgitation.	28/52	Good	Nil	–	+	+	—	—	g.	D.	Infant not viable.
11	E. R.	2	34	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	Twins.
12	M. G.	5	28	Mitral Stenosis and Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	
13	M. Y.	4	43	Mitral Stenosis.	Full	Good	Nil	–	+	+	—	—	g.	g.	
14	E. W.	3	34	Mitral Stenosis and Regurgitation.	36/52	Poor	Nil	–	–	+	Bag & Version	Slight	g.	g.	
15	F. H.	3	27	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	
16	R. C.	4	24	Mitral Regurgitation.	Full	Good	Trace	+	–	–	—	—	g.	g.	
17	A. A.	4	24	Mitral Stenosis and Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	[death 19th day.
18	H. H.	6	40	Mitral Regurgitation.	28/52	Bad	No note	+	–	–	—	Bad	D.	D.	Precip. labour. Increas. cardiac incompetence,
19	M. B.	9	41	Mitral Stenosis and Regurgitation.	Full	Good	Trace	–	+	+	—	Slight	g.	g.	Slight and occasional dyspnœa during puer-
20	M. M.	3	38	Mitral Regurgitation.	Full	Bad	4/5ths	+	–	–	—	Bad	g.	g.	Cyanosis and dyspnœa after. [perium.
21	A. R.	10	42	Mitral Regurgitation and Aortic.	38/52	Good	Nil	+	–	–	Induction	—	g.	g.	
22	L. G.	7	47	Mitral Regurgitation.	Full	Good	Trace	+	–	–	—	—	g.	g.	
23	F. R.	10	36	Mitral Stenosis.	35/52	Good	Nil	+	–	–	—	—	g.	g.	
24	L. V.	7	37	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	
25	E. S.	3	39	Mitral Regurgitation.	24/52	Poor	Nil	+	–	–	—	—	g.	D.	Not viable.
26	S. R.	11	39	Mitral Regurgitation.	Full	Good	Nil	+	–	–	—	—	g.	g.	
27	R. W.	4	30	Mitral Stenosis.	Full	Poor	No note	–	+	+	—	—	g.	g.	One attack of dyspnœa in puerperium.
28	K. H.	4	39	Mitral Stenosis.	26/52	Good	No note	+	–	–	—	Slight	g.	D.	Not viable.
29	J. W.	3	24	Mitral Stenosis.	Full	Good	Nil	+	–	–	—	—	g.	g.	
30	C. G.	5	28	Mitral Stenosis and Regurgitation.	36/52	Bad	Solid	+	–	–	—	Mod.	g.	g.	
31	M. H.	7	39	Mitral Regurgitation.	Full	Good	Nil								

A Note on Adenoma of the Labium.*

By Herbert Williamson, M.A., M.B.

My object in writing this note is to draw attention to a rare form of tumour of the labium, which is liable to be mistaken clinically for squamous-celled carcinoma.

Three years ago Dr. Champneys removed from the labium of a lady a small tumour which he regarded as an epithelioma. Microscopic examination showed that the growth was composed of glandular tissue, and that the ulceration of the surface was of a simple, not of a malignant, type. Dr. F. W. Andrewes pronounced the growth an adenoma; but, from the irregular appearance of some of the epithelial masses, I felt doubtful of the diagnosis, and thought the nodule might be a secondary deposit from a glandular cancer primary in some other part of the body. I have waited three years before reporting this case, and as the lady is still in good health, I am convinced that Dr. Andrewes was right, and that I was wrong. I am indebted to Dr. Champneys for the clinical notes of the case and for his kindness in investigating the after-history.

The patient was an unmarried lady of 62; for two and a half years she had suffered from a slight blood-stained vaginal discharge, and on one occasion, a year before consulting Dr. Champneys, there had been a sudden hæmorrhage, the quantity of blood lost being sufficient to soak one diaper. At the free edge of the right labium majus, near its anterior extremity and at some distance from the orifice of the right vulvo-vaginal gland duct, was a small growth equal in size to a split-pea. The growth was soft, pink in colour and ulcerated on the surface, and it bled very readily on touch. No enlargement of the inguinal glands could be detected.

The growth was removed in March, 1903, and at the present time (June, 1906) the patient's medical attendant reports that she is in good health and that no recurrence has taken place.

Microscopical examination. The tumour was attached to the labium by a broad base composed of delicate connective tissue, in which are embedded groups of large, thin-walled vessels. The base

* Read at a meeting of the Obstetrical Society of London, July 4th, 1906.

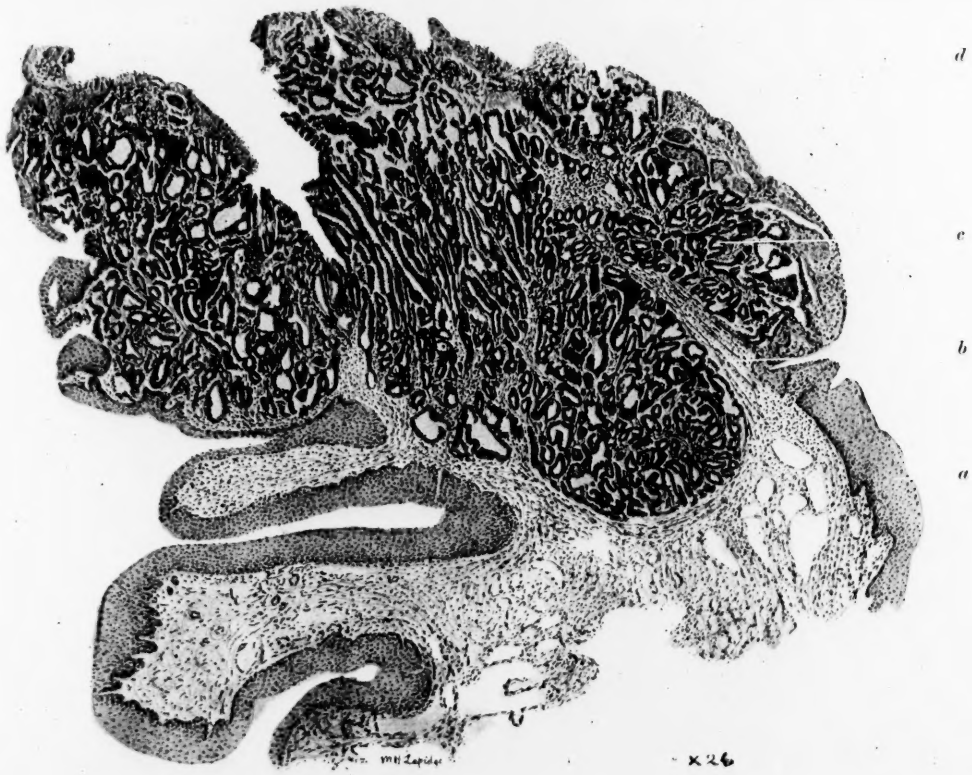
of attachment and the proximal part of the tumour are covered by several layers of stratified squamous epithelium; the distal portion evidently possessed originally a similar epithelial covering; traces of it still remain, but, for the most part, it has been destroyed by a process of ulceration. The glandular tissue which constitutes the greater part of the growth is partially sub-divided into lobules by fibrous septa, and is marked off from the subjacent connective tissue by an incomplete capsule of concentrically arranged fibres.

The tumour consists of irregular glandular formations embedded in a delicate loose-meshed connective-tissue stroma. The gland acini and ducts vary in size and form; some are lined by a single layer of tall columnar cells resting upon a well-marked basement membrane and possessing a vesicular nucleus situate near the base of the cell; some of the acini are dilated and form cystic spaces. The whole growth, however, is not of this simple type; many of the lumina are lined by two or three layers, the deeper cells rounded, the more superficial columnar, but the basement membrane is still preserved intact. Some of the tubes show irregular branchings and anastomoses between the branches of adjacent acini; there are also present, embedded in the stroma, masses and solid columns of epithelial cells; these are for the most part sharply defined from the surrounding connective-tissue and possess characters similar to those of the cells which line the lumina. It was the presence of these irregular columns and masses which made me suspicious that the tumour was possibly of a malignant nature. The stroma at the more superficial part of the tumour is infiltrated with inflammatory cells.

I have succeeded in finding records of three similar growths; two cases are reported by Pick and one by Schickele. As far as I am aware, no description of these tumours is to be found in English literature.

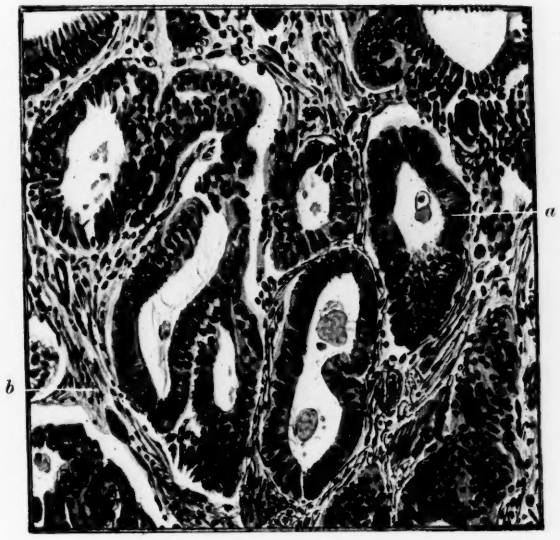
Case I. (L. Pick). An unmarried woman, 40 years of age, had noticed for the last two years a small tumour on the posterior part of the left labium majus. Shortly before she sought advice two others had appeared, one on the right labium and the third on the left labium.

On examination a small red-coloured tumour the size of a bean was seen on the inner aspect of the right labium majus, near its free border, at the level of the urethral orifice; a second, somewhat larger, was situated on the left labium, close to the opening of Bartholin's duct, and near to this was a third smaller one. All the



Adenoma of Labium—Low Power.

a. Surface covering of squamous epithelium. *b*. Incomplete fibrous capsule. *c*. Adenomatous growth. *d*. Ulcerated area of surface.



Adenoma of Labium—High Power.

a. Glandular structure. *b.* Connective tissue stroma.

tumours presented similar characters; they were hard, the surface was smooth and covered with hair, they could be separated from the skin, and could be moved easily on the deeper structures.

The tumours were removed, and, at the time the case was reported, no recurrence had taken place.

The microscopical appearances were almost identical with those of the tumour I have described.

Case II. (L. Pick). The patient was a woman, 45 years of age, the subject of arthritis deformans affecting both hands and feet, and also of prolapsus uteri. A single small tumour was situated in the posterior part of the right labium majus and possessed characters very similar to those of the previous case. Its surface was smooth; the growth could be separated from the skin and was freely movable upon the deeper tissues.

The microscopical appearances differed from those in the previous case only in certain minor points: the gland ducts were extremely tortuous; there was no cystic dilatation; there was no definite capsule, and the growth was not lobulated.

Case III. (Schickele). The patient was 35 years of age, and had borne three children. Like the patient in the previous case, she suffered from prolapsus uteri. In the upper part of the right labium minus was a nodule the size of a cherry-stone. It was soft and movable on the underlying structures, but attached to the skin.

The microscopic examination showed that the tumour, which possessed a glandular structure, was composed of epithelial ducts, but the ducts nowhere reached the surface. Immediately beneath the skin were small masses of round cells.

The pathology of adenomatous growths of the vulva is not easy to understand; two theories have been suggested, one by Schickele, the other by Pick. Schickele believes that the tumours arise in connection with abnormal remains of the Wolffian duct. He bases his view upon the resemblance which the growths bear to the kidney, the testicle, and to Wolffian structures in general. To my mind, there are two objections to this theory:—first, the varying situations which the growths occupy; sometimes they are found in the anterior, sometimes in the posterior part of the labium majus, and sometimes in the labium minus. Had they developed in connection with an embryonal structure one would expect them to occupy a more constant position. Secondly, as far as I am aware, no one has ever found Wolffian remains in the labium, and it is almost

impossible to understand how they could reach that situation, for in animals which possess a persistent Wolffian duct this structure terminates in the vagina, not in the labium.

Pick regards these growths as adenomata arising in connection with sweat-glands, and proposes for them the name "Midradenomata." He bases this opinion upon the resemblance between the growths and the normal sweat-glands, and upon the fact that a few cases of adenomata described in connection with sweat-glands in other parts of the body possess many points in common with these tumours. I have cut sections from five specimens of the labium minus taken from the post mortem room, and have failed to find sweat-glands in any of them; they are, however, sometimes to be found in this situation, and have been described and figured by Clarence Webster in his study of the nerve-endings in the labia minora.

I have already remarked that when I examined the section I was in grave doubt as to whether the tumour was a secondary nodule of glandular cancer. At that time Pick's paper had not been published, but on reading it later I was interested to find that he had been struck by the close resemblance of these growths to malignant adenomata. "Indeed," he writes, "at first I took them to be metastases of such a growth." Schickele still regards them as probably of a malignant nature.

The case I have recorded has been under observation longer than any of the three previously published, and therefore, after waiting three years, I offer it as a contribution to the natural history of these growths.

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1. Pick. "Ueber eine besondere Form nodulärer Adenome der Vulva." *Archiv für Gynäkol.*, 1904, p. 347.

2. Schickele. "Weitere Beiträge zur Lehre mesonephrischen Tumoren." *Hegar's Beiträge zur Geburts. und Gynäkol.*, 1902, Bd. 6, Ht. 3.

3. Webster. "The nerve-endings in the labia minora and clitoris." *Edin. Med. Journal*, vol. xxxvii., p. 35.

SELECT CLINICAL REPORTS.

(Under this heading are recorded, singly or in groups, cases to which a special interest attaches either from their unusual character or from being, in a special sense, typical examples of their class.)

I.

Case of Chorionepithelioma following Vesicular Mole.

By J. K. Kelly, M.D.,
Physician for Diseases of Women, Royal Infirmary, Glasgow.

On the 9th of March, 1906, I was asked by Dr. Brownridge, of Paisley, to see his patient, Mrs. S., aged 38, the mother of three children. She was about three months pregnant, and for six weeks had suffered from sickness and vomiting and red discharge from the vagina. This discharge lately had been at times very profuse, so much so as to cause faintness on several occasions. One specially severe hæmorrhage had taken place just before I saw her.

The uterus was larger than was accounted for by the duration of pregnancy, and on examination *per vaginam* it had a peculiar feeling as if its surface was irregular from innumerable small nodules. Although there had been no discharge of vesicles in the blood, I expressed the opinion that it was a case of vesicular mole, and advised that if it were not expelled within a day or two the patient should be sent to hospital for its removal.

She was admitted to Ward 30 on the 14th of March, when the following report was made:—

Mrs. S., æt. 38, married 14 years; 3 children, the last 4 years ago; no abortions; labours normal. Menstruation 4-weekly, lasting 2 or 3 days with slight pain occasionally. Is well nourished, but pale. Mammæ enlarged, secreting; areolæ dark. Patient complains chiefly of sickness, vomiting and weakness. Her last menstrual period was in the second week of December. She felt very well for 5 weeks after that, when she lost her appeite. In the sixth week she had a slight red discharge, and sickness and vomiting started. The discharge lasted 2 days, then stopped for 2 days, and then came on again for a day. Sickness and vomiting became so troublesome that she sent

for her doctor. He gave her powders which helped her, but slight sickness and vomiting have continued ever since. Discharge again started 10 days ago, and was very profuse for 2 days. It gradually ceased, but 3 days later it again came on for 1 day. Last night she had profuse discharge again. No clots have been passed. There has been no pain. But for the discharge and the sickness she would feel well.

Physical Examination. The abdomen is rather large, moderately thick-walled. The hypogastrium is occupied by a mass reaching about two finger breadths from the umbilicus and from about the level of the middle of right Poupart to 1½ inches from the left A.S. spine. This mass is slightly movable from side to side, and is rather more prominent to the right of the middle line.

Per Vaginam. The cervix is low, considerably enlarged and expanded, soft and irregular on the surface. The mass felt in the abdomen is the enlarged uterus, over the lower part of which arterial pulsation is very marked. There is no pain on examination.

On the evening of the 15th, four laminaria tents were introduced into the cervix, and on the morning of the 16th the uterus was emptied of a large vesicular mole, over the lower part of which a mass of blood-clot was attached. The interior of the uterus was carefully curetted, and then packed with iodoform gauze. The patient was extremely collapsed, both before and after the operation, and had two saline transfusions and frequent hypodermic injections of strychnine for the following two days. The gauze was removed next morning, and there was no further bleeding.

On the 22nd, under local cocaine, several small cysts—like vesicles of the mole—were excised from the lower part of the posterior and lateral wall of the vagina.

The patient went home well on the 26th, nothing malignant having been discovered either in the vesicular mass removed from the uterus or in the vesicles excised from the vagina. Her doctor, however, was informed that after vesicular mole there was sometimes a development of chorionepithelioma, and he was asked to report to us as to the progress of the case.

She was re-admitted to the ward on April 23rd with the following report:—Since leaving the ward, 4 weeks ago. she has felt very weak, only sitting up for an hour at a time, and then feeling quite exhausted. A fortnight ago, there was a faint, straw-coloured discharge from the vagina, which was always darker coloured in the morning. This continued more or less for a week. On the 13th of April a very profuse hæmorrhage set in, and continued from 2 p.m.

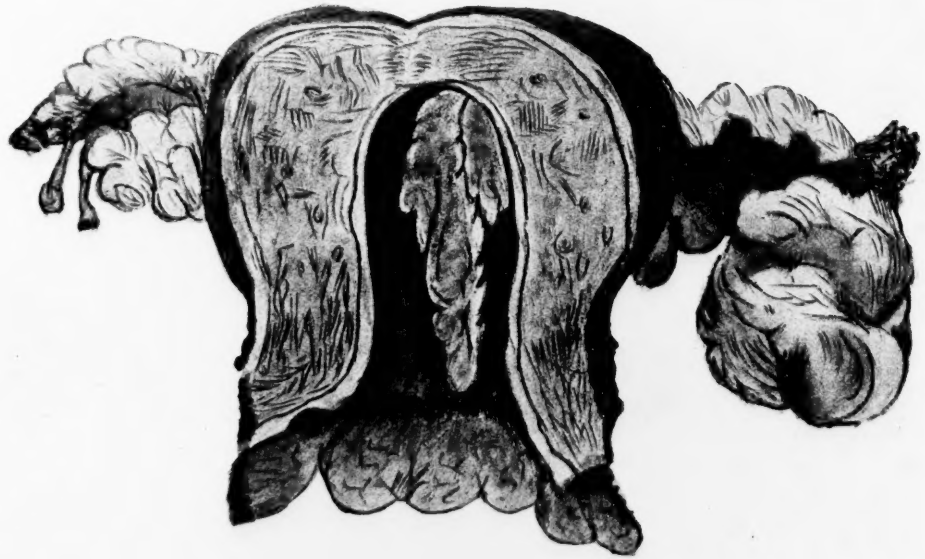

FIG. 1.—Uterus opened along anterior surface showing tumour depending from left angle; the upper part pure chorionepithelioma, the lower part blood clot.

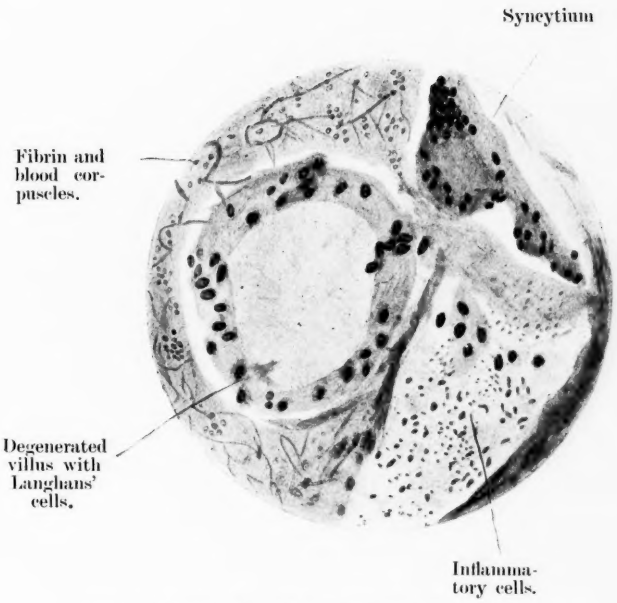

FIG. 2.—Section from Mole. Objective 1/2, Ocular 6.

FIG. 3.—Section from Tumour. Objective ⅛, Ocular 6.

FIG. 4.—Section from Tumour. Objective 1, Ocular 10.

till 10 p.m. It continued in less amount for 3 days, then stopped for a day, and again recommenced and continued in moderate amount till the 20th, with small clots in the discharge. On the 20th she had a very profuse hæmorrhage with large cysts. Her doctor found her almost pulseless, and plugged the vagina. She felt faint, but did not faint altogether. On the 21st a smaller plug was put in as the bleeding was not so severe, and the plug was left out on the 22nd. The bleeding returned in the early morning of the 23rd, and her case was considered hopeless, when I saw her and, after firmly packing the upper part of the vagina, had her removed in an ambulance to the Royal Infirmary. There was no further bleeding, and on the morning of the 24th I performed an abdominal hysterectomy, removing ovaries and tubes as well. She made a smooth recovery, and left the ward on the 14th of May; up to the present time (June 25th) she continues well.

Immediately after the operation the uterus was laid open by a longitudinal incision along the middle of the anterior wall, and a small polypoid tumour was found projecting from the neighbourhood of the left tubal opening, and from the lower end of this mass a small blood-clot hung down into the internal os (Fig 1). On microscopic examination this tumour showed the characteristic structure of chorionepithelioma (Figs. 2, 3 and 4).

During the removal also an orange-sized cyst of the right ovary was ruptured and gave vent to pale, straw-coloured fluid. The wall of this cyst showed the ordinary structure of the corpus luteum.

This case illustrates the connection, which is now so well known, between vesicular mole and chorionepithelioma, and also the connection between cyst of the corpus luteum and vesicular mole which is insisted upon by L. Fraenkel and others.

As to the concurrence of cysts of the ovary with chorion-epithelioma, I may refer to a case which I had in the ward last year (to be published shortly in the *Glasgow Medical Journal* as part of the Transactions of the Glasgow Pathological and Clinical Society, to which it was shown). In this case, which was one of extraordinary malignancy, from a small chorionepithelioma in the anterior uterine wall there were metastases in the vagina, heart, spleen, lungs, kidney and brain, and the ovaries, which were also infected, were both cystic (each equal in size to a Jaffa orange).

II.

Case of Round-celled Sarcoma of the Ovary Penetrating the Uterine Wall and Presenting in the Vagina.

By J. K. Kelly, M.D.,

Physician for Diseases of Women, Royal Infirmary, Glasgow.

F.C., æt. 53, virgin, admitted into the Glasgow Royal Infirmary on the 15th of June, 1906, complaining of fœtid watery discharge from the vagina since December, 1905. The menopause had not yet occurred. Menstruation was never excessive, but when the patient was young it was accompanied by pain.

She had her usual period in October, and in December this thin discharge began, and has been constant since. In January she again menstruated, but it has not recurred since. The discharge is brownish at times, but usually almost colourless. After a vaginal examination it is of a dark brown colour. It has a foul, musty smell, but she says she has formerly perceived as bad an odour after a usual monthly period. The odour varies in intensity.

She experienced no pain till the last week of April. Since then she has had constant grinding pain across the lower abdomen, and worse in the left iliac region than elsewhere. She has also a dull pain of the same "grinding" character across the small of the back, which began 10 days ago. The pain in the abdomen is at its worst during the day, between 10 a.m. and 4 p.m., and is easier in the evenings. Of late there has been some pain on micturition, but the bowels have been regular.

The patient is very pale, and rather inclined to stoutness, though she says she is much thinner than she was. The hair is grey, almost white. Except for the extreme pallor she does not give the impression of illness.

The thoracic organs seem normal except that the second cardiac sound is much accentuated and the first sound scarcely heard. The urine is normal.

The abdomen is large, and marked by striæ, which extend also over the upper thighs. On palpation the lower part of the abdomen is found occupied by a very hard rounded mass extending to the umbilicus, slightly movable, both laterally and vertically, and almost insensitive to pressure.

Per Vaginam. The cervix is spread out all round the os, the latter presenting a rounded opening with smooth borders just large enough to admit the tip of the finger, which, when pressed up, touches a softish, irregular mass apparently occupying the interior of the uterus. There is no bleeding on examination, but the discharge is very fœtid. The mass felt in the abdomen seems to be formed by the uterus distended by a tumour.

About a week before admission she was seen by Dr. G. Balfour Marshall, who found a soft mass protruding from the cervix. This he broke down and removed with his fingers, and sent to be examined microscopically. It proved to be a round-celled sarcoma.

The diagnosis naturally was "uterine myoma undergoing sarcomatous degeneration," but the absence of any history of menorrhagia made the previous existence of a myoma rather doubtful. A hysterectomy was proposed.

After admission the offensive discharge continued. The evening temperature was about 100·2°, and the morning about normal. The pain was moderate. She had no rigors, but perspired very freely.

On the 20th of June abdominal section was performed. The tumour was found to fill the whole posterior part of the pelvis, the uterus being spread along its anterior surface. It evidently originated from the left ovary, was slightly nodulated on the surface, and was attached to the bowel both above and behind and on the left side. Shortly after leaving the angle of the uterus the left Fallopian tube became suddenly distended, and all its outer part formed a tortuous sausage-shaped hydrosalpinx lying over the left upper part of the tumour. Whilst manipulating the tumour a small part of it broke away, and the bowel was slightly separated from its upper part, but there was no hæmorrhage. It was considered inadvisable to proceed with the operation, and the abdominal wound was closed. It was evidently a case of sarcoma of the left ovary, which had penetrated the posterior uterine wall, and examination of the small piece which had broken off showed exactly the same round-celled sarcoma as the pieces obtained from the cervix.

The case was clearly hopeless, but I did not anticipate the course that it took. For over 24 hours the patient was unusually well, had very little sickness or pain, passed urine naturally, and perspired freely as she had done before the operation. When I saw her the following morning her temperature was 99·6°, and her pulse 98. By midnight, however, the temperature had reached 102·2°, and the pulse 120. She had vomited several times, had passed flatus, and had perspired as usual.

Early on the next day the temperature was 102·6°, pulse 126, and though the patient felt no distress, her breathing was short and rapid, her skin was clammy, the pallor of her face was more marked, and it was evident that toxæmia was rapidly developing. The temperature rose to 105° at 4 p.m., and she died at 5-50, having retained consciousness and being free from pain to the last.

At the post mortem examination the liver was found extremely fatty. The gall-bladder contained thick fluid bile. The heart, spleen and kidneys were normal. The lungs were slightly congested. A large mass, equal to the size of two fists occupied the lower left half of the abdomen with the uterus firmly adherent to its anterior surface and with the left Fallopian tube distended. The sigmoid flexure of the colon and the rectum were inseparable from the tumour, and were removed along with it. The tumour was soft, and was breaking down in its lower part which perforated the posterior uterine wall just above the cervix. The uterus itself seemed perfectly normal.

On section the mass presented an encephaloid appearance with darker and lighter portions. Unfortunately no further examination was made, as, owing to a misunderstanding, the museum attendant put away the specimen.

This case is the only one I have seen in which a sarcoma of the ovary penetrated the uterine wall. Such an occurrence might quite reasonably be expected, but it must be very rare, and is worthy of record.

The rapidly fatal termination also is noteworthy. There was no peritonitis, no renal complication, no cardiac condition to account for it. Nor was it likely to be due to septic absorption from the necrosing lower part of the tumour, though that had evidently been slowly poisoning the patient for some time, as evidenced by the temperature and perspiration. I am inclined to regard it as due to the manipulation of the tumour and to the breach of the surface caused by the separation of the bowel from it and by the breaking away of the small portion mentioned above. But in an ordinary ovarian cyst or myoma or even cancerous ovary all this may occur without any bad result. I would suggest that there was a rapid absorption from the tumour itself, either of some toxin or of active cellular elements, and here again it must be regretted that a fuller examination and consideration of the specimen were not obtained.

III.

Six Cases of Myomectomy During Pregnancy.

By E. T. Thring, F.R.C.S. (Eng.), L.R.C.P. (Lond.),

Surgeon to the Prince Alfred Hospital and to the Royal Hospital for Women, Sydney, New South Wales.

Case I. Mrs. W., æt. 29; married one year. Previous history unimportant. First seen October 23rd, 1901. Menstrual periods regular up to May, 1901; then ceased. Patient believes she is pregnant, and says that she has a "lump" on right side of abdomen, which causes some pain and inconvenience.

On examination. Heart, lungs, etc., normal; uterus pregnant about six months; to the right of the uterus, and high up, is a hard or tense mass, equal in size to a large orange, evidently attached to or near the uterus, movable to some extent but not freely.

Diagnosis. Either a tense ovarian cyst or a uterine fibroid.

Operation, October 28th, 1901. Incision at outer border of right rectus over tumour, which was a pedunculated uterine fibroid attached to the right anterior part of the fundus by a pedicle about an inch and a quarter in diameter. The tumour was removed, two curved incisions being made, one on each side of the pedicle, thus forming two short flaps; vessels picked up and ligatured with fine catgut; edges of flaps joined by continuous catgut sutures; abdomen closed in layers. Patient made an uninterrupted recovery, and at term was delivered, without trouble, of a living child. No stretching of abdominal cicatrix.

Case II. Mrs. S., æt. 39; married twelve years; four children, the last one being $3\frac{1}{2}$ years old; one miscarriage $2\frac{1}{2}$ years after last confinement. Menstruation regular until $3\frac{1}{2}$ months before admission to hospital, on November 27th, 1903. Some frequency of micturition, and pain beforehand. Patient complains of "a lump in her side," noticed about $2\frac{1}{2}$ months before; pain in it during past two months, also a somewhat rapid increase in size.

On examination. Uterus evidently pregnant between three and four months; fundus forward, rather high up and lying to the

right of the middle line; in the pelvis, to the left and behind, was a mass attached to the uterus, moving with it, and about equal in size to the body of the pregnant uterus. The mass could not be lifted out of the pelvis, and would obviously obstruct delivery of fœtus through the pelvic outlet.

Operation, December 1st, 1903. Under ether, incision through left rectus sheath, slightly to the left of the middle line. Fibro-myoma found wedged in pelvis, attached to the lower segment of the uterine body and upper part of the cervix behind and to the left, pushing the uterus upwards, forwards, and to the right. Incision made over tumour, which was shelled out; bleeding controlled by catgut ligatures and two rows of buried catgut sutures, which also closed raw surfaces left by enucleation of tumour; edges of incision brought together by continuous catgut suture; abdomen closed in layers. Patient made an uneventful recovery, and was duly delivered at term of a living child. No stretching of abdominal cicatrix.

Case III. Mrs. J., age 24. Admitted to Prince Alfred Hospital June 3rd, 1904. Married five months; no previous pregnancy. Menstrual periods regular every four weeks, the last one early in March, 1904, nothing since. Shortly before this, *i.e.* February 1904, patient noticed a swelling in the lower abdomen; no pain.

On examination. Uterus enlarged, pushed upwards and to the left, the cervix uteri being on a level with the left pubic spine; the pelvis filled by a soft, smooth tumour, upon which the uterus was lying, and to which it was attached.

Diagnosis. Either a large, soft fibro-myoma or an adherent ovarian cyst; former more probable.

Operation, June 7th, 1904. Under ether; incision through inner margin of right rectus sheath; large fibroid lying between layers of broad ligament on right side; uterus pushed up and to the left; about 3½ months pregnant; incision made through broad ligament, parallel to right round ligament, and between it and right Fallopian tube; tumour shelled out. It was attached to the right posterior part of the cervix uteri, the feeding vessels being in close relationship to the uterine artery and right ureter. The vessels were ligatured with catgut, and the tumour was removed. Two layers of buried catgut suture closed the incision into cervix uteri, and a continuous catgut suture closed the line of incision in the broad ligament. The ovaries and Fallopian tubes were normal,

and were not interfered with. The abdomen was closed in layers. The patient made an uninterrupted recovery, and the pregnancy ended in a normal labour at full time. No yielding of abdominal cicatrix.

Case iv. Mrs. B., æt 40 years; married. No children or miscarriages. Is now, *i.e.* January 20th, 1904, pregnant 6½ months. For about the last four weeks patient had had epigastric pain and discomfort, also tenderness on pressure; these are increasing. Temperature varies from 98·4°F. to 101°F. Menstruation before pregnancy was regular about every four weeks.

On examination. Patient a big, rather stout woman; heart and lungs normal; abdomen showed a large central tumour, reaching quite up to the costal angle; tender on pressure at the upper part. The uterus was pregnant, and there was a tumour which could not be separated from the uterus, and which touched the under surface of the liver.

Diagnosis. Either an intra-mural fibro-myoma of the uterus or a hyatid tumour of the liver, probably the former.

Operation, January 27th, 1904. An abdominal incision above the umbilicus, through sheath of right rectus; tumour found to be a fibro-myoma, which was enucleated through an incision through the fundus and anterior surface of uterus. The deeper portion of the tumour was close down to the uterine cavity. Some vessels were tied, and the uterine wound was closed by layers of catgut sutures. Abdominal incision closed in layers; no drain. The uterus was manipulated as little as possible apart from the incision and the enucleation of the tumour. The ovaries and Fallopian tubes being normal were not interfered with. The patient aborted about 36 hours after operation. Fœtus, placenta, and membranes came away easily. As the uterus did not contract well, and there was a slight rise of temperature, up to 100°F., a curetting, under an anæsthetic, was done on the fifth day. Recovery after this was uneventful. On July 21st, 1904, *i.e.* nearly six months after operation, the patient was examined. Uterus a little larger than the usual normal non-pregnant uterus, freely movable; no pain or tenderness. There has been no menstrual period since the commencement of the pregnancy, *i.e.* July 17th, 1904.

Specimen. A fibro-myoma about equal in size to an ostrich's egg, but somewhat flattened. On section, there were seen in several places signs of commencing cystic degeneration, and the microscope showed necrotic changes to have commenced.

Case v. Mrs. E., æt. 34; married; three children. The patient is rather stupid, and, as a consequence, her previous history is not very trustworthy. Was first seen by me on October 10th, 1904. Says she menstruated 10 days before this, not having previously done so for six weeks; before that, she was "regular."

On examination. Abdomen showed signs of previous pregnancy. There is a central tumour rising out of the pelvis, a little above the pelvic brim. Bi-manual examination showed pelvis to be occupied by a round, firm mass impacted below sacral promontory; could not be lifted; body of uterus apparently pushed upwards and forwards in middle line; somewhat enlarged, but not diagnosed as being pregnant.

Operation, October 11th, 1904. Under anæsthesia a sound was gently passed into uterus, cavity nearly 5 inches; incision made through right rectus sheath; pregnant uterus came into view; fibromyoma filling pelvis and pushing uterus upwards and forwards. With some difficulty the tumour was lifted so far as to allow an incision to be made over it. Bleeding from large venous sinuses was free. The tumour was rapidly enucleated, and the bleeding was controlled by catgut ligatures and sutures in layers. The tumour was attached by its principal vessels to the lower uterine segment, and the upper part of the cervix. The ovaries and Fallopian tubes were left undisturbed, as they were normal.

In this case, as there had been more manipulation required than usual, and the patient was unhealthy-looking and "flabby," a gauze drain was placed from Douglas's pouch into the vagina, so as to allow serum to drain away.

For one week all went well and then certain untoward events occurred. The temperature had been normal, and the patient was apparently making good progress, when on the eighth day the whole abdominal incision opened up, and coils of small gut presented. An anæsthetic was given, and the wound was again closed by through-and-through sutures of silkworm gut. There was no evidence of peritonitis, and the opportunity was taken to examine the line of suture in the uterus, where the myoma had been. This was soundly healed. The edges of the abdominal wound, which came apart, showed scarcely any signs of reparative action, only a few small patches of lymph. Subsequently to the second closure of the abdomen there was an irregular temperature, which has, however, now (November 1st, 1904), *i.e.* just one month after the original

operation, fallen to normal. The abdominal wound is soundly healed and the patient looks well. There has been no abortion.

Case vi. Mrs. F. G., æt. 38 years; nullipara.

History. Up to present illness was unimportant. Patient was seen by Dr. Abbott November 20th, 1904. She was then complaining of pain, tenderness and discomfort in the epigastric region. Temperature varying from 98·6°F. to 101°F. On examination Dr. Abbott found the uterus pregnant between six and seven months. The fœtus was alive. There was a definite tumour to be felt above the umbilicus, and apparently connected with the fundus uteri. It was tender when manipulated. November 24th, 1904, the patient was seen by Dr. Abbott and myself, and we agreed that the condition was one of pregnancy of about the above-mentioned duration complicated by a uterine fibroid, which was not pedunculated, but intra-mural, and was apparently undergoing degenerative changes, most probably necrotic in character.

It was decided to operate with the intention of removing the tumour, and, if possible, not interfering with the pregnancy. The patient was removed to a private hospital.

Operation, November 29th, 1904. Abdominal incision to right of middle line, above umbilicus and over the tumour. The fundus uteri was exposed, and an incision made through the uterine wall down to the tumour, which was placed deeply. The uterus was steadied from the outside, and the tumour was enucleated, the greatest care being taken to handle the uterus as little as possible. At one point the mucous membrane lining the uterine cavity was exposed. The uterine incision was sutured in three layers with chromicised catgut. Abdominal incision closed in layers. No drainage.

The patient made an uninterrupted recovery, and returned home at the end of three and a half weeks.

The confinement took place at "full time" during the first week of March, 1905. It was uneventful, and the patient is now (April 10th, 1905) up and about. The infant was normal and well nourished.

Pathological Report.

By Dr. Smith, *Pathologist to the Prince Alfred Hospital.*

Uterine Tumour submitted by Dr. Thring. The tumour was a hard ovoid fibrous-looking mass about equal in size to a large orange. It gave the typical macroscopic appearance of a fibro-

myoma, but on section the cut surface showed four distinct areas of somewhat darker colour than the surrounding whitish tissue. These areas were discrete, about the average size of a threepenny-piece and of a fairly dark brown colour. They were somewhat softer than the other parts of the cut surface. They gave no macroscopic evidence of a hæmorrhagic origin.

With due precautions cultures were taken from each area, on to agar and blood agar, and into broth, and incubated. No growth whatsoever appeared. Each area was fixed and imbedded in paraffin and sections stained for organisms and with routine stains. No bacteria whatsoever were discovered on exhaustive examination.

Histologically the tumour was found to be a fibro-myoma, the fibrous element very largely predominating, the fibrous intersections being very fine and exceedingly numerous. Scattered throughout the sections were small areas of breaking-down tissue, apparently of necrotic origin. No signs of hæmorrhage were discovered and the sections showed absolutely no evidence of an infective process of any kind whatsoever.

Thus the bacteriological examination with the secondary histological examination gave a definitely negative result as to an infective origin of the patches described. They were apparently of necrotic origin.

The above cases are interesting from several points of view. All except Case V. have been seen or have been heard from recently and are well. There was no mortality. In only one instance (Case IV.) did premature labour result, and it seems scarcely probable that the pregnancy would have gone on to term even if no operation had been performed, as the symptoms of illness, *i.e.* pain, local tenderness and a rise of temperature, were daily becoming more marked.

In Cases I., II., III., and VI., the pregnancies went on to full time, and labour is known to have been normal and uneventful. Had the tumours which occupied the pelvis in Cases II. and III. been then *in situ*, this would not have been possible. Some obstetrical operation would have been required, and even then the tumour would have had to be reckoned with after delivery.

Case VI. is especially interesting, because of the advanced stage of the pregnancy, with which the operation in no way interfered, and also because of the careful bacteriological examination of the specimen. This gave absolutely negative results, and shows that the necrotic changes which had taken place were not due to the

presence of organisms, although accompanied by rise of temperature. Although by no means an advocate for the immediate removal of all uterine fibroids which may be found upon examination, I do believe that in *most* cases removal is the right course to follow, particularly if there are indications of degenerative change or excessive bleeding. But more especially does this seem to hold good where pregnancy occurs, the tumour being impacted in the pelvis and consequently offering an obstruction to delivery *per vias naturales*.

A report of the earlier cases was read before the New South Wales Branch of the British Medical Association, in November, 1904.

IV.

Three Cases of Post Partum Ovariotomy.*

By J. A. C. Kynoch, M.B., F.R.C.P., F.R.C.S. (Ed.),
Professor of Obstetrics and Gynæcology, University of St. Andrews.

It is generally agreed that when an ovarian tumour is found complicating pregnancy it should be immediately removed. Delay in operating increases the risk to the mother, and the chances of disturbing the course of pregnancy are likewise less the earlier the condition is recognized and treated. The literature on this subject is almost endless, as shown, for example, by a glance at the index of the *Zentralblatt für Gynäkologie* during the past ten years. What strikes one on a study of those contributions is the endeavour to break the record of previous results and to prove how safely ovariotomy can be performed during pregnancy. Without going into details of the literature it may be mentioned that McKerron has collected 480 cases, with a mortality of 5 per cent., which could be reduced to 2 per cent. by excluding fatal cases where other causes might account for the unfavourable result. Those figures far surpass the results in cases of non-interference, where, for example, in a collection of 720 the mortality was as high as 21 per cent. Figures might also be quoted to prove the beneficial results to the child by early operation.

Of four cases of ovariotomy during pregnancy, I have operated on one at the third month, on two at the fourth month, and on one at the seventh month. In the first three abdominal ovariotomy was performed, and in the case at the seventh month a dermoid tumour was removed by vaginal section. The course of pregnancy was not disturbed in any of the cases, all going on to full time. Torsion of the pedicle was present in the three abdominal cases, but only in one did it give rise to acute symptoms demanding immediate operation. In the other cases the unusual size of the abdomen led to a correct

* Read at a meeting of the Edinburgh Obstetrical Society, June 13th, 1906.

diagnosis. In the case at the seventh month attention was directed to the condition by pelvic discomfort. The tumour was a dermoid in two of the cases; in one it was a multilocular and in the other a papillomatous cyst. In the case where there was extreme torsion of the pedicle, all the usual symptoms of that condition were present, accounted for by extreme hæmorrhage into the cavity and cyst wall. I have not met with a case of ovarian tumour complicating actual labour, a condition which has recently been discussed here by Dr. Haultain. My object in bringing this short communication before the Society is to report three cases of ovariotomy during the puerperium, a subject which has received but scanty attention in comparison with the question of ovariotomy during pregnancy and labour.

Although it is undeniable that pregnancy, labour, and the puerperium may be gone through without any mishap in the presence of an ovarian cyst, yet the dangers associated with axial rotation of the pedicle, rupture, and suppuration of the cyst are specially prone to occur during the puerperium, thereby increasing the risk to the patient. This being so, the advantages of ante partum ovariotomy are obvious. The fact of labour being one of the commonest causes of suppuration of the cyst, whether the infection be through genital tears or of intestinal origin, accounts for the frequency of the condition during the puerperium. Gelter has reported 21 cases where, in spite of normal labours, suppuration of the cyst has occurred and led to fatal peritonitis. Gottschalk has emphasized the importance of the bacillus coli as setting up infective changes, resulting in the formation of pus and gas within the cyst. The chances of axial rotation are likewise greater during the puerperium because of the laxity of the abdominal wall permitting greater mobility of the cyst, and lowering of the abdominal pressure at the time of the emptying of the uterus. As the result of the frequency with which infective changes are set up in an ovarian cyst during the puerperium, most of these cases have been mistaken for puerperal sepsis, an error resulting in non-recognition of the true cause of the febrile symptoms and the almost invariable death of the patient. Aust Lawrence reported ten such cases sent to hospital, diagnosed as puerperal peritonitis.

The following are the notes of my cases:—

Case 1. J.S., aged 19, was delivered normally of a full-time healthy child on 20th Feb., 1902. After the birth it was noticed by

the midwives in attendance that the abdominal swelling had not disappeared; this was supposed to be accounted for by the presence of a second child. Their patience diminishing and their anxiety increasing, they sent the case to hospital. On admission, the patient stated that she had noticed a swelling of the abdomen some months before pregnancy had occurred, but throughout the pregnancy she had had no discomfort. The temperature was 99°, pulse 100. The abdomen was uniformly distended by a cystic swelling, the circumference at the umbilicus being 41 inches. There was no œdema, and except for deficient urinary output the various systems were normal. The patient remained quite comfortable till the twenty-second day after confinement, when the temperature rose to 104°, with pulse 136. On account of her unsatisfactory condition ovariotomy was performed, and a large multilocular cyst arising from the left ovary was removed. The main cyst was full of fœtid pus, and the pedicle was twisted, but not very tightly. The operation was completed in the usual way and the abdominal cavity irrigated with saline solution. The patient made a straightforward recovery.

TABLE OF TEMPERATURE AND PULSE FROM FIFTH DAY OF PUERPERIUM. CASE I.

1902.	Temperature.	Pulse.	1902.	Temperature.	Pulse.
Feb. 25, M.	98·6°	92	Mar. 8, M.	98·6°	100
E.	100·8°	104	E.	101·5°	104
,, 26, M.	99·7°	102	,, 9, M.	97·0°	98
E.	100.4°	100	E.	100·1°	108
,, 27, M.	98·6°	96	,, 10, M.	98·6°	100
E.	100·6°	92	E.	99·0°	110
,, 28, M.	99·5°	90	,, 11, M.	98·6°	100
E.	100·4°	92	E.	101·0°	104
Mar. 1. M.	98·6°	92	,, 12, M.	100·4°	102
E.	100·3°	104	E.	102·0°	112
,, 2, M.	98·6°	102	,, 13, M.	100·0°	120
E.	100·6°	110	E.	104·0°	136
,, 3, M.	98·0°	100	,, 14, M.	102·0°	122
E.	100·8°	88	E.	103·6°	120
,, 4, M.	98·4°	96	,, 15, M.	103·0°	120
E.	99·5°	96			operation
,, 5, M.	97·3°	80	E.	101·0°	120
E.	98·6°	84	,, 16. M.	101·0°	116
,, 6, M.	97·0°	84	E.	99·0°	112
E.	99·0°	98	,, 17, M.	97·0°	106
,, 7, M.	98·0°	84	E.	98·6°	102
E.	101·9°	108			

Case II. Mrs. McD., 24 years of age, was delivered of a full-time healthy child, 14 days before admission to hospital. This was her second confinement, and the child was born before assistance reached her. She remained in bed for eight days, when she tried to resume her domestic duties, but as she had severe pain in the lower part of the abdomen she was compelled to return to bed and summon medical aid. On admission to hospital she looked very ill. The temperature was 103°, and the pulse 136. There was a tender, cystic swelling extending in the middle line to one inch above the umbilicus. The urinary and other systems were normal. Immediate operation showed that the swelling was a right-sided multilocular ovarian cyst with extreme torsion of the pedicle, the twist involving the uterus. As a consequence the cyst wall was gangrenous in places, and in others showed well-marked hæmorrhages. The operation was completed in the usual way, and recovery was satisfactory.

Table of Temperature and Pulse from Day of Admission. Case II.

1905.	Temperature.	Pulse.	1905.	Temperature.	Pulse.
Dec. 20, M.			Dec. 24, M.	98·0°	84
E.	103·7°	136	E.	98·2°	84
,, 21, M.	100·0°	120 Ovari-	,, 25, M.	98·3°	90
E.	101·8°	136 otomy	E.	98·6°	88
,, 22, M.	98·0°	108	,, 26, M.	98·2°	88
E.	99·5°	104	E.	98·6°	88
,, 23, M.	98·5°	104	,, 27, M.	98·2°	88
E.	98·2°	92	E.	98·6°	84

Case III. Mrs. S., aged 37, was confined of her third child on the 13th of June, 1905. She had thought herself unusually large during her pregnancy, but otherwise made no complaints until after her confinement. I saw her on the 24th of January on account of the persistence of an abdominal swelling, which had been diagnosed (by her own doctor) during her pregnancy as an ovarian cyst. (Operation had been proposed at the time, but declined.) When I first saw her she was perfectly comfortable, with a normal temperature and pulse. The abdomen was uniformly distended to the size of a full-time pregnancy by a cystic swelling. The physical signs pointed to the swelling being a unilocular ovarian cyst. Her condition not giving rise to any special discomfort I deferred operating till the end of the fourth week after her confinement, when I removed a unilocular cyst of the left ovary, unaccompanied by adhesions

or any signs of inflammatory change. The patient's recovery was satisfactory.

The first two cases—one a case of suppuration and the other of gangrene of the cyst—show how necessary immediate operation becomes in a puerperal woman known to have an ovarian tumour, and exhibiting unfavourable symptoms. The third case shows that immediate operation is not, however, always imperative, and that, in the absence of unfavourable symptoms, surgical measures may, perhaps with advantage, be delayed until the patient has completely recovered from the effects of her confinement.

REVIEW OF CURRENT LITERATURE.

OBSTETRICS.

Confinements during Artificial Half-conscious Sleep (Dämmerschlaf).

GAUSS. *Archiv. für Gynäkol.* Band lxxviii., Heft 3.

GAUSS reports upon 500 confinements in the obstetric department of the University Hospital at Freiburg, where skopolomin-morphia injections were used. These drugs did not create complete anæsthesia, yet prevented the feeling of acute pain. During the course of his experiments Gauss succeeded in producing a degree of semi-consciousness in which the patient moaned during the pains, and gave rational answers, but dozed in the intervals between the pains, and when she woke up finally could not recollect either pains or conversation; she could not even remember the fact that the baby had been born, although she seemed to have been fully aware of it at the time. Gauss found that different women reacted in a different degree to the drugs, and required larger or smaller doses. A very few women slept soundly all through the confinement, and another small group (7 cases) reacted in the opposite way—became violently excited, but this condition subsided immediately after labour, without leaving a trace in the patient's memory. The action of skopolomin-morphia is not sufficient to relax the abdominal wall, so when operative measures (high forceps, etc.) were required, additional anæsthetics had to be given, and Gauss found that all the common anæsthetics—chloroform, gas, ethyl chloride—agreed well after skopolomin-morphia, and that the period of excitement generally accompanying their administration was absent.

Gauss generally used solutions prepared by Merck, and kept the two drugs separate: sol. skopol. 0·03 per cent., and sol. morph. mur. 1 per cent. As a rule he began with an injection of skopol. hydrobrom. 0·00045 to 0·0006 grm., and morph. mur. 0·01 grm., which mostly acted within $\frac{3}{4}$—3 hours. If not successful, a second and weaker dose was given and repeated every 2—4 hours, but in de-

creasing doses. In three cases he extended the semi-sleep over 50—57 hours without producing any ill-effects. The largest collective quantity for one patient was skopol. 0·0031 grm., and morph. 0·0475 grm., given over a period of 48 hours. The patient's surroundings should be kept very quiet. When by experience he had learnt the right doses the efficiency of the pains was never diminished, and in comparing his tables with those of other accoucheurs the duration of labour under skopolomin-morphia does not exceed the normal. Although skopolomin-morphia diminishes the secretion of glands and mucous membranes, it has no lasting or bad effects on the secretion of milk. Skopolomin-morphia produces no lasting ill-effects on the child; occasionally the child was born in a curious condition—taking one deep breath, accompanied by a loud scream; and then remaining motionless with a fair pulse, and giving shallow, gasping respiratory movements. At first this condition caused a good deal of anxiety, but it was soon found that even when no efforts at resuscitation were made the child recovered satisfactorily, and that superficial stimulation of the skin soon established regular breathing. The quickest and most efficacious reaction was produced by rhythmical massage of the heart. The oligopnœic condition in the baby was not noticed when there had been sufficient time to induce semi-sleep gradually, as then the doses could be carefully calculated. Eighteen of the 500 children were born asphyxiated; for this no explanation could be found at the time, but as none of these infants exhibited any of the symptoms of intoxication mentioned above it would be rash to attribute the condition to the action of the injections. Five of these cases did not recover, and satisfactory causes for the fatal issue were found post mortem. The subsequent mortality of babies born during skopolomin-morphia treatment did not differ from that of other children.

Contra-indications for the treatment are: Inefficient primary pains, general debility, high temperature, specific fevers, acute anæmia, and any conditions tending to obscure the clinical picture. The disturbance of consciousness in eclampsia is no contra-indication, and the injections have proved beneficial in producing a pacifying action. The University Hospitals of Graz, Jena, Giessen, Budapest and Klagenfurt have used skopolomin-morphia with equally satisfactory results.

H. T. Hicks.

Cæsarean Section for Imminent Rupture of the Uterus.

Potocki. *Annales de Gynécol. et d'Obstét.*, May 1906.

Since rupture of the uterus is one of the most grave accidents of midwifery, and is so frequently accompanied with fatal results, the author insists on the recognition of its premonitory symptoms and signs, and urges that by timely intervention its occurrence can be avoided. Recently cases of this sort have been reported both by Pinard and the author.

A very interesting case is reported in Potocki's papers of a woman of 24, pregnant for the third time. On being admitted to the maternity hospital the uterus was found acutely anteverted, and the head above the brim in the right iliac fossa. The pelvis was generally contracted, and of a rickety type. As the cervix dilated well the membranes were artificially ruptured but the head would not descend. The uterine contractions became very strong and were accompanied with violent pain. A hot bath was given but with no result. The patient was very restless and the cervix and vulva became œdematous. When Potocki saw the case later, the uterus was tense, almost wooden in hardness, and of a curious shape. In the region of the neck of the child was a more or less marked depression below which the bulging of the fœtal head was felt. Besides this Potocki remarked a very curious soft swelling which he at first took to be the bladder. On a catheter being passed only a few drops of urine escaped, and the swelling remained as before. The tumour was found to be the much thickened and œdematous lower uterine segment. By vaginal examination the cervix was much thickened, very œdematous, and so rigid that even under chloroform the finger could not be passed between it and the fœtal head without risk of rupture.

On these grounds forceps or the use of the basiotribe was out of the question. Symphysiotomy was not deemed advisable on account of the great contraction of the œdematous cervix upon the fœtal head, and because rapid evacuation of the uterus seemed necessary. Craniotomy was not justified since the child was alive and well. Cæsarean section was therefore performed with good results. The uterine wound was closed with catgut sutures in two layers. The abdomen was drained by gauze which was removed three days afterwards. The child weighed 3·075 kgms., the head was very much ossified and hard with marked asymmetry, and a deep depression

existed on the left parietal bone. The author justifies his treatment of the case and thinks that Cæsarean section is indicated in all cases of *threatened* rupture of the uterus except hydrocephalus, which of course should be treated by simple perforation.

C. Hubert Roberts.

Recent Inversion of the Uterus Successfully treated by de Ribes's Bag.

Mantel (Paul), reported by Pinard (A.). *Annales de Gynécol. et d'Obstét.*, May 1906.

The case in question occurred in a multipara of 26. Her first confinement was ended by forceps, and the second by normal means. Her third child was delivered by forceps, the labour lasting 20 hours. The period of expulsion occupied seven hours. Acute inversion of the uterus occurred during the period of delivery, probably owing to traction on the cord. The placenta, which was situated at the fundus, had become detached, but the membranes were very adherent at its periphery. These were separated, and the whole uterus was easily replaced. On bimanual palpation the fundus was found in its proper position in the abdomen. Five days afterwards, as the patient was passing urine, she felt something give way, but no grave symptoms of collapse or bleeding ensued. On examination the next day the uterus was found to be again completely inverted into the vagina. Manual reposition was now found to be impossible, and after careful disinfection of the fundus and its surroundings, de Ribes's bag was inserted into the vagina. Forty-eight hours after the bag was removed, the inversion was found to have completely disappeared. The bag was re-introduced and kept *in situ* for eight days longer. The patient recovered completely. The interest of this observation is that an acute inversion was *reproduced* four days after its occurrence and replacement. The cause was intra-abdominal pressure during the act of micturition. Cases of acute inversion are very rare, and Pinard remarks that he himself has only seen one case in 60,000 deliveries. The most frequent cause seems to be traction on the cord, especially if the placenta be inserted *exactly* at the fundus. In a case reported by Queirel, which was absolutely spontaneous, this insertion of the placenta was also noted. Other predisposing causes are primary or secondary uterine inertia, and thus inversion is liable to occur in forceps deliveries. Shortness of the cord, absolute or re-

lative, may be a factor, and in rare cases intra- or extra-uterine tumours.

Pinard would divide cases of inversion into three groups:—(a) *immediate*, (*i.e.*, produced during the third stage or during the period of delivery; (b) *recent*, existing some hours or days after; and (c) *chronic*, existing weeks or months afterwards.

Regarding treatment in these cases, Pinard advises immediate manual reduction in the first variety, separating the placenta before attempting replacement of the fundus. The patient is then to be kept at absolute rest, avoiding all effort or straining, and if the inversion recurs the introduction of de Ribes's bag.

For the second variety, when sloughing is not threatened, disinfection and manual reposition should be attempted. If this fail, de Ribes's bag should be employed. This is better treatment than plugging the vagina or the use of stem pessaries. Various sizes of de Ribes's bag may be necessary for complete success. Lastly, should the uterus be found sloughing, reduction is out of the question, and surgical interference is indicated. Operation may also be necessary in a large number of cases of chronic inversion.

C. Hubert Roberts.

Surgical Treatment of Puerperal Pyæmia.

Trendelenburg (F.) (Leipzig). *Journ. Amer. Med. Assoc.*, July, 1906.

In a recent address in the section of surgery and anatomy of the American Association (June 1906), Trendelenburg, in referring to advances in surgery brought about by the use of antiseptics, states that surgical pyæmia is only met with at present in two forms, as result of infection from the middle ear or from the uterus. He compares the two conditions, and asks why the same line of treatment should not be followed in the latter, as in the former, namely by clearing out and ligaturing thrombosed veins.

In both conditions there is a thrombo-phlebitis with a resulting diffuse inflammation or localized abscess in the region of the thrombus.

In referring to the high mortality in puerperal pyæmia, Trendelenburg discusses the question of surgical interference along these lines, and believes that this method of treatment will be used largely in the future to the exclusion of hysterectomy He reports

a case, in which he ligatured the right internal iliac vein, which was thrombosed, as also the right ovarian vein, with recovery, and refers to several other successful cases so treated.

He recommends ligature of the ovarian and uterine veins in all cases of acute, and in chronic pyæmia.

Two methods may be used, transperitoneal or extraperitoneal, but in the former there is always risk of peritoneal infection. In the extraperitoneal method, the incision is made as for ligature of the iliac artery.

E. Scott Carmichael.

The Unreliability of Curettage in Cases of Retained Placenta after Abortion.

Lepage (G.). *Compt. rend. de la Soc. d'Obstét., de Gynécol. et de Pediat. de Paris.* Vol. viii., No. 5, May, 1906.

The author discusses at length the relative advantages of curettage and digital removal of the placenta when it is retained after abortion.

He gives as the arguments generally advanced in favour of curettage:—

1. The curette is more easily introduced than the fingers, especially in the first two months, when it is almost impossible to introduce the latter.

2. In septic cases it obviates soiling the fingers of the operator.

The arguments against it are:—

1. It is a dangerous instrument which, if not skilfully handled, may perforate the uterine wall.

2. The instrument has no sensation, and consequently portions of placental tissue may be left behind.

On the whole he considers the arguments against the use of the curette, especially when used alone, stronger than those in its favour, and lays especial stress on the danger of incomplete removal of the placenta.

He quotes a considerable number of cases emphasizing this point: two of the most interesting of which, perhaps, are cases where the uterus was curetted with a view to procuring abortion at about three months, yet the patients were delivered of living children at full term.

On the other hand he quotes two cases where digital examination of the uterus was made and still fragments were left behind. These

he attributes to imperfect dilatation of the cervix and consequent difficulty in carrying out the exploration.

He also emphasizes the fact that it is only after thorough digital examination that one can say definitely that the uterus is empty in cases complicated by other conditions.

HAROLD CLIFFORD.

GYNÆCOLOGY.

The Influence of the Uterine Nerves upon the Atony of the Non-puerperal Uterus.

FELLNER (L.) (Franzensbad). *Zentralbl. f. Gynäk.*, 1906, No. 26.

AFTER obtaining unquestionable results from experimental studies on animals, the author considers that he is able to demonstrate the nerves which produce atony of the non-puerperal uterus. In a former paper the author showed that the *nervus erigens* (a branch of the sacral plexus terminating in the hypogastric plexus) is the motor nerve for the longitudinal muscles and the inhibiting nerve for the circular muscles of the rectum, and that the hypogastric nerve (a pair of nerves springing from the ganglion mesentericum posterior, and ending in the hypogastric plexus) carries motor fibres for the circular muscles and inhibiting fibres for the longitudinal muscles of the rectum. The innervation of the two parts of the uterus—cervix and corpus—is distinct, and there is an antagonistic action between the two. The corpus uteri is governed by the same motor and inhibitory nerves as the rectum—erigens and hypogastric, whereas in the cervix an opposite action is carried out by the same nerves. Both dilatation and curetting set up a mechanical irritation, and a prolonged irritation causes a nerve fatigue and consequent laxity of the muscles. This condition may be produced by the application of a tent for 24 hours, but also in a shorter time by a more intense repeated irritation, such as the introduction of metal dilators.

The author (like Schaeffer) distinguishes two degrees—the stage of a balloonlike distension with a diminished but not altogether absent tone, and a stage of entire relaxation—atony and paralysis of the uterus. In the first stage the uterus appears as a balloon, and forms a bag with thickened walls, especially in the sagittal diameter, while the horns either hang in the cavity or outside it like loose bags. In the second stage the uterus is uniformly enlarged, the walls are thinned out and a slack bag is thus produced. The first stage is due

to dilatation, the cervical ganglion being stimulated and becoming paralysed. This paralysis is communicated to the hypogastric nerve, and the result is paralysis of the circular muscle of the uterine body. But as the nervus erigens is not connected with the cervical ganglion but with the ganglion situated in the fundus, it remains intact, and the longitudinal muscles of the corpus uteri keep their tone, hence the thickening of the wall. In addition the nervus erigens is the vaso-dilator of the uterus, and this action is all the greater, as the action of the vaso-constricting hypogastric nerve is missing, hence the vessels of the uterus are enlarged and tense. The second stage—paralysis—only sets in with curetting when the ganglion in the fundus becomes paralysed. This paralysis is communicated to the nervus erigens which is in contact with this ganglion. Now the longitudinal muscles also are paralysed, and the uterus forms this uniformly dilated bag with slack walls. Naturally this atony and paralysis of the uterus are only produced when pathological changes in the uterus or adnexa or other general diseases act as a predisposing cause. The author suggests the terms "hypotony and atony" respectively for the two conditions.

H. T. Hicks.

The Histology of Adeno-carcinoma of the Uterine Fundus.

Offergeld. *Archiv für Gynäkol.* Band lxxviii., Heft 2, p. 289.

Offergeld in an exhaustive article upon the histology of adeno-carcinoma in the uterine fundus, discusses in detail its different histological forms. He looks upon the horny, squamous-celled carcinoma, which is sometimes found in the uterine fundus, as the result of a change from the cylindrical cell to a squamous type. To such a change Veit has applied the term ichthyosis uteri or epidermization. He records 15 cases of adeno-carcinoma, and comes to the following conclusions:—There is no evidence to show that pre-existing inflammatory changes of the genital organs or gynæcological treatment prevent the occurrence of adeno-carcinoma. Carcinoma of the body must, if possible, be removed as soon as recognized, as early metastases are found in the neighbouring glands and the ovaries. All the different varieties of carcinoma are equally malignant. The adeno-carcinoma of the uterus is generally a mixture of several varieties. It penetrates into the muscle, but to a lesser extent than carcinoma glandulare simplex. During the process of degeneration in the foci, sarcoma-like effects are produced.

Some specific changes in the tissue are noticed at an early date, viz., degeneration and absorption of the membrana propria and a dipping in of the epithelium, alteration in the differentiation of the plasma and nuclei of the cells; further, an abnormal quantity of mitoses with a large percentage of pathological forms and giant cells. The regeneration of carcinomatous epithelium is irregular and rarely complete, and often assumes an alveolar character. Malignant degeneration of a clinically benign epithelial tumour is not proved, and is improbable. The secretion of an adeno-carcinoma is profuse, and consists of fibrin and mucus, and most likely also of fibrogenous and albuminoid bodies which produce a toxic action upon the tissues, especially the elastic elements, the capsule of the glands and the whole organism (cachexia).

In order to establish a correct diagnosis, a good many pieces of endometrium should be removed for examination by curetting. Frequently a glandular endometritis co-exists with early glandular carcinoma, and the exploratory curetting may have to be done repeatedly. The metastases, occurring in distal parts of the urino-genital system during primary carcinoma of the body, must be looked upon as carried by the lymphatics or blood-vessels. There is no proof that secondary deposits occur by direct infection or implantation.

As regards the ætiology, Offergeld supports Cohnheim's theory. He thinks the tissues are able to produce antitoxins which prevent further growth of the abnormally present cells. When the formation of alexin ceases, either altogether or for the time being, embryonic remains are able to develop, but they do this in an abnormal direction, because the altered tissues cannot supply them with the necessary material for the morphological and physiological development of their cells. An alteration in the position of blastomeres and cells is facilitated by various causes, and the large expanse of the uro-genital system in the embryo explains the frequency of carcinoma of the uterus (10 per cent. of all carcinomata).

Offergeld thinks that the character of adeno-carcinoma should be looked upon as a retrogressive process invading the neighbouring tissues, and that the proliferation of the cells is secondary.

H. T. Hicks.

The Use of Oxygen in Gynæcology.

Thiriar (J.). *Revue de Gynécol. et de Chir. Abd.*, June, 1906.

Professor Thiriar, of Brussels, has long advocated the employment of oxygen in septic infections due to anærobic organisms, and par-

ticularly the gas bacillus. More recently he has maintained that though oxygen possesses no bactericidal power over the ærobic organisms, its action does diminish their virulence and destroy their toxins, while at the same time it increases the resistance of the individual.

The present article deals with the use of the oxygen method in gynæcological work: first, as a means of preventing septic infections in both vaginal and abdominal operations; secondly, as a cure for these infections should they occur; and finally, as a method of treatment in certain gynæcological diseases or affections.

Thiriar maintains that there is no method of cleansing the vagina preparatory to operation equal to a prolonged disinfection by pure oxygen. The current must be kept up for several hours, and apparently this is always well tolerated by the patient, and presents no difficulties in technique. In regard to abdominal sections, wherever there is any suspicion of fouling of the peritoneum, as for example when, in removing a pyosalpinx, the contents have escaped into the pelvis, he establishes a continuous current in the abdomen, sometimes for as much as three or four days. Two tubes are necessary, forming a siphon, and the current should never be very strong. It should be regulated by the tolerance of the patient; if too strong it will cause pain, while if too weak it may lead to ballooning of the belly. It should be mentioned that Thiriar does not neglect the ordinary precautions of the surgeon, *i.e.* careful cleansing of the peritoneum, and occasional drainage; he only adds to these ordinary precautions the special disinfection by the oxygen current, and he claims that this oxygen method produces a drainage much more efficacious, and more lasting, and that its use hinders and prevents post-operative gynæcological infections.

Should infection of the peritoneum occur from any cause, and apparently whatever the organism may be, Thiriar once more pins his faith on oxygen as the most reliable therapeutic agent. He mentions the various substances which have been used in the endeavour to increase the resistance of the peritoneum, and to provoke an abundant leucocytosis (toxins, albumose, nucleic acid, etc.), but he declares that both laboratory experience and clinical results point to the employment of oxygen, "which acts at one and the same time on the microbes, on their toxins, and on the means of defence of the organism."

As regards its use in gynæcological affections it will be understood that Thiriar employs it in all septic infections of the external genital organs (particularly one may mention suppuration in Bartholin's

gland) and in vaginitis. It is admitted that the gonococcus shows a certain resistance to oxygen, but the current will have a cleansing effect as regards the associated microbes, and thus render it easy to attack the gonococcus itself with tincture of iodine, nitrate of silver, or other therapeutic agent.

Passing to the internal genital organs we come to this somewhat remarkable statement, "inflammations of the uterus have benefited so much by this method, that curettage, so frequently employed formerly, has become nearly a rarity in my service." This statement is a little modified later, where it is stated that the oxygen method gives somewhat different results according to the character of the metritis, and that in certain cases it should be an adjunct to curettage, while in others it should be followed by microbicidal injections. A short description of the technique employed in the intra-uterine application of oxygen follows this statement of its value, and Thiriar adds that if a feeble current is used, with a double tube, there is no danger of entrance into veins, even after a recent curetting.

The use of the method in relation to pelvic suppuration has already been mentioned in this abstract, and need not be again referred to. It should be said in conclusion that no attempt has been made here to appraise the value of the method, the only wish being to record the claims made for it by Professor Thiriar, who has now practised it for some years, and has already published several memoirs on the subject.

R. Hamilton Bell.

REPORTS OF SOCIETIES.

BRITISH GYNÆCOLOGICAL SOCIETY.

Meeting held on July 12th, 1906, Mr. F. B. JESSETT, *the President, in the Chair.*

Dr. JERVOIS AARONS showed a modified Hollow Ring Pessary, filled with a mixture of gelatine and glycerine, used in retroflexion with prolapsed ovaries.

Dr. CHARLES MAUNSELL showed the following specimens:—(1) *Uterus and Adnexa*, removed by Wertheim's operation for malignant disease of the cervix. The glands were removed up to the kidneys, and, although enlarged, no evidence of malignant disease was found. The ureters were easily found by tilting forward the ovaries, when they readily showed through the peritoneum. The patient had urinary leakage on the tenth day, which healed up in a few weeks after the bladder was washed out regularly. (2) Specimen of *Carcinoma of the right Ovary*, from a single patient, aged 25, who complained of a swelling in the abdomen. On opening the abdomen some ascitic fluid was found, and there were adhesions between the tumour and surrounding structures, also between the liver and anterior wall of the abdomen. The left ovary contained a small cyst, which was not malignant. The patient had increased in weight, and so far there is no evidence of metastasis or recurrence. (3) *Myoma of the Cervix*, from a patient, aged 47, who had metrorrhagia for two years. The tumour bled profusely when touched, so that vaginal plugging was necessary. On drawing the tumour down it was found to be attached by a narrow pedicle, which was snipped through with scissors. The patient recovered satisfactorily. (4) A *Tubo-ovarian Abscess* of one side and *Salpingitis* of the other tube, removed from a multipara, aged 24, fifteen months previously. The patient continued well for some months, and then complained of severe pain and vaginal discharge. The uterus was removed, and found to be in a condition of suppuration. The patient made a good recovery. (5) Two large *Tubo-ovarian Abscesses* of gonorrhœal origin, removed from a patient, aged 23, who had no children, and had been married about a year. She complained of vaginal discharge with pelvic pain. Before operation she had two attacks of sudden severe pain, and when seen was collapsed. On opening the abdomen pus welled up from the peritoneum, and both tubal abscesses were found ruptured. Two incisions were made, one above and the other below the umbilicus; the abdominal cavity was washed out and drained with gauze. The patient made an uninterrupted recovery. (6) Two *Tubo-ovarian Abscesses* which ruptured before operation. The same line of treatment was adopted as in the former case, but the patient died a few days afterwards.

Dr. MACNAUGHTON JONES showed a specimen of *Hydatidiform Mole*, removed from a patient, aged 20, who had been married eighteen months and had one still-born child at term. She considered herself to be four

months pregnant, but had a hæmorrhagic discharge for some time. The uterus was emptied, and the specimen shows degenerated decidual tissue and chorionic villi, showing hyperplasia of Langhans' layer with syncytial buds. As a precautionary measure, the uterus was curetted two months afterwards.

Dr. JAMES OLIVER read a paper, entitled,

A STUDY OF HYDATIDIFORM MOLE, WITH THE RECORDS OF THREE TYPICAL CASES.

After mentioning the theories of menstruation, and expressing himself in favour of the view that there is no destruction of tissue, he briefly described the anatomical relationship between the allantois and chorion. The pathogenesis dates from the embryonic period between the third and sixth weeks. The interaction of allantois and chorion is at first harmonious. This interaction may be disturbed by the vigorous local growth associated with the formation of the placenta, and may induce a perversion of the function of the allantois, which may prejudicially influence the vessels and more or less obliterate them, so that a mole may result. The functionally deranged placental, and even non-placental, villi may excite such reaction in the uterine wall, that a more or less intimate union may be established between embryonic and maternal tissue, so that the morbid process connected with this union may be injurious, not only during gestation, but also after abortion. Hydatidiform mole may be found at any time during reproductive life. The symptoms were then detailed. The association of amenorrhœa with the presence of a mole raises an interesting point, as in cases of intra-uterine death it cannot be assumed that the suspension of menstruation can be solely due to the presence of a perished ovum in the uterus. The derangement is probably due to some disturbance in the metabolism of the uterus, for the abortive or aberrant product of conception attracts to itself and absorbs nutriment which is elaborated and practically secreted by the endometrium. The demand made thus upon the uterus is very different from that made by an ordinary neoplasm, and induces such an alteration in its physico-chemical state generally that not only is menstruation held in abeyance, but the irritability is so impaired that it may tolerate for an indefinite time the presence of a vesicular mole in its cavity. A uterus of a smaller size than ought to be, considering the suspected duration of pregnancy, is an important phenomenon. Three cases of molar pregnancy were then detailed, and a discussion followed.

REVIEWS OF RECENT BOOKS.

THE TECHNIQUE OF VAGINAL OPERATIONS. By E. Wertheim and Th. Micholitsch. With 138 illustrations. Leipzig: S. Hirzel.

The authors have achieved their aim and object in presenting to the beginner a systematic description of each vaginal-abdominal operation, illustrated, step by step, by the aid of photography. For photographic purposes the narrowness and depth of the vaginal region were effectively overcome by symphysiotomy. This ingenious device enlarged and levelled the otherwise deeply placed, important anatomical structures. Anatomical details within the field of operation have come out so clearly in the illustrations that an easy shallow, rather than an awkward depth, is suggested to the eye by almost every picture.

The general introduction deals with the opening of the abdomen by the vaginal route, *i.e.*, with anterior and posterior vaginal cœliotomy, in the normal pelvis. Eleven photographs, excellently reproduced, illustrate this section.

The advantages of anterior cœliotomy are precisely stated. In opening the utero-vesical pouch during this operation, a proper appraisement is attached to the "sharp separation" of the bladder (Plate 3) in preference to the "blunt working" between the bladder and the cervix uteri, as the manipulations of the latter so often inconveniently raise the desired peritoneal fold. The transverse incision across the vaginal vault at the bladder border is advocated together with the final incomplete closure with a small central opening for gauze drainage.

Posterior vaginal cœliotomy, the simpler operation, affords relatively less accessibility of the uterus and adnexa, and greater effort is needed to deliver the internal genitalia for inspection. As an alternative to their delivery, a view of these organs *in situ*, by ventroscopy, after von Ott's method of using retractors with blades carrying small incandescent lamps for illumination, is mentioned in connection with posterior vaginal cœliotomy. The presentation and delivery of the uterus and its appendages, after either anterior or posterior vaginal cœliotomy, are effected by the use of sharp hooks, bullet-forceps and small spatulæ, displayed in Plates 12 to 26. During the withdrawal and replacement of the organs, inconvenience, difficulty and danger are to be averted by the provision of an adequately large opening in the vaginal roof; by gentleness in manipulation; by the substitution of the forefinger for the traction-hook, if the latter tears the tissues; by the selection of light and delicate hooks and forceps, and by their application at the right places for traction. Heavy volsellæ are to be shunned.

A special part (A) of the work is devoted to the series of vaginal operations with retention of the uterus, including (1) vaginal shortening of the round ligaments; (2) vaginal fixation of the uterus; (3) intra-vaginal fixation of the uterus; (4) shortening of the utero-sacral ligaments; (5) vaginal myomectomy; (6) vaginal operations on the tubes; (7) vaginal ovariotomy.

For the shortening of the round ligaments by the vaginal route (Plates 30—32) (in the operative treatment of retroflexion of the uterus) a relatively favourable position is claimed on the grounds that if the loops formed are large and well-secured by silk button-sutures the forward and upward position of the uterus is permanently secured; and that in the case of a heavy uterine body the loops made by the shortening may be additionally stitched in the vagina.

Vaginal fixation of the uterus (Plate 33), intra-vaginal fixation of the uterus (Plates 34–35) and shortening of the utero-sacral ligaments (Plate 36), as operative procedures, are amply explained, but judiciously limited by definitely expressed restrictions as to the applicability of each operation, as to the site and kind of suture and as to the extent of excision or mutilation. Landau's resection of the uterus amounts to more or less total extirpation of the uterine body. In gross uterine prolapse with large cystocele or rectocele, the whole, or the resected part, of the body of the uterus is used as the carrier of the bladder or the rectum as the case may require; for a large cystocele an anterior cœliotomy and colporrhaphy enable the operator to bury the whole, or the resected part, of the body of the uterus under the anterior vaginal lining as the support of the bladder; and for a large rectocele after posterior cœliotomy and colporrhaphy, similarly, the whole, or the resected part, of the body of the uterus is interposed between the principal layers of the recto-vaginal septum as the carrier of the rectum.

Vaginal myomectomy with or without *morcellement* (Plates 37—55) receives the attention which this important operation deserves.

In vaginal operations on the tubes, resections and somato-plastic procedures being excluded, the operator is advised to secure the tubal attachment at the uterine end, and gradually separate the tubal connections to the uterus and broad ligament. In this way tubal sacs and tubal pregnancies can be treated provided no severe adhesions are present. As the vaginal operation reduces the risk of peritoneal infection, puncture or incision of the tube is rather to be encouraged than avoided. Only gentle manipulations are to be employed. Anterior cœliotomy is to be preferred.

Vaginal ovariotomy, on similar lines, necessitates an adequate presentation of the tumour, and the main purpose undoubtedly is to render the pedicle quite accessible. If the pedicle be short, anterior cœliotomy, and if the pedicle be long, posterior cœliotomy is the easier operation. Plates 56—77 illustrate the steps of these operations. The complications and contra-indications are correctly cited in the text. In borderland cases, the precaution of making a preliminary digital exploration of the tumour and its surroundings, through a posterior cœliotomy incision, establishes the choice of the abdominal route, if required, for the extirpation of the tumour.

The special part (B) contains exhaustive illustrations (Plates 75—111) and a carefully compiled text concerning extirpation of the uterus, the operation commencing either (*a*) from the corpus or (*b*) from the collum for either cancerous or fibroid growths. Variations from a set operation are approved. The attention of the beginner is specially directed to the general advantage of withdrawing the body of the uterus in front and during deep or wide dissections of the parametrium of having the pars vesicalis of the ureter pushed well upwards and forwards or prepared by exposure through a specially higher anterior cœliotomy incision. Wide and high pelvic dissections are aided by the incisions of Schuchardt and Staude (Plate 103).

The special part (C) devoted to the extirpation of the uterus and adnexa contains all the essential information on operative technique, copiously illustrated in Plates 112—138.

The work is the first of its kind; an exhaustive and systematic description, and the best available technique have been upheld throughout its preparation.

Photography has not lent colour effects to guide the operator's study of the plates. Allusion to the pale pink muscular fibres of the bladder as a danger signal in its dissection or separation has been omitted from the text.

Inflation of the lower bowel is also omitted, yet sometimes it is a great help to the practical surgeon working amidst deep and dense pelvic matting.

Concerning the arrest of hæmorrhage during vaginal-abdominal operations, the authors' summary will be found attractive and trustworthy. There is one defect in the use of ligatures; they require to be tightly tied on a secure base, a practical difficulty which calls for a remedy. There is no evidence that lacing a continuous suture by short steps, advancing a little ahead of the scissors, along the vascular tissues, prior to their division, is not simpler and easier of application and more free from the danger of slipping; knots on the laced suture are tied only after the tissues have been severed and lost their tension, one knot after every three or four loops of suture is a common average along the broad ligaments, the para-vaginal or the para-rectal tissues.

The need of drainage after vaginal-abdominal operations is regulated by the authors on the soundest general surgical grounds; gauze is more applicable than any other material as a drain; where an unusually large quantity of gauze has been placed its gradual withdrawal by daily intermissions, beginning after the second day, is a wise precaution against omental or intestinal prolapse.

The methods in secondary injury with which the work closes are briefly alluded to as, firstly, preventative, and, secondly, reparative. Repair can usually be carried out at once if the injury be recognised at the time.

A unique addition has been made by the authors to the literature of operative gynæcology.

A System of Gynæcology. By many writers. Edited by T. C. Allbutt W. S. Playfair and T. W. Eden. Second Edition. Pp. 949. London: Macmillan and Co., 1906. Price 25s. net.

The appearance of a new edition of Allbutt and Playfair's System is an event of the first importance in the gynæcological world. The aim of the editors has been, with the assistance of a staff of selected specialist writers of eminence, to produce a work that shall serve as an authoritative representation of the present position of this department of medicine in Great Britain. Continental and American authors may be expected to look in the System for the latest and best-considered achievements of British gynæcological pathology, medicine and surgery, and it is therefore a matter of personal interest to every practising specialist in the diseases of women that the work should have been well done by the men best qualified for the purpose. The first edition was made up of articles of very unequal merit; along with a number of papers of the highest excellence were many of average and a few of distinctly poor quality. Such variety is, of course, inevitable in the many writer system of book production; in the present edition the attempt has been made, with some degree of success, to level up the quality.

In the ten years that have passed since the first edition appeared death has been busy in the ranks of the contributors, and new men have had to be called in to fill up the gaps. The original responsible editor, Dr. Playfair, has gone; and to fill his chair Dr. Eden has been selected by Prof. Allbutt. No better choice could have been made; to a wide and profound knowledge of his subject, and to considerable practice as a worker and a writer, has been added valuable experience gained in the positions, first of assistant Editor, and afterwards of Editor, of this Journal. Of the other writers, two of the greatest, Knowsley Thornton and Greig Smith, have passed away, and left our ranks the poorer. In their places are found Professors J. W. Taylor and H. R. Spencer, whose names are a sufficient guarantee of the authority and value of the papers to which their names are attached.

Since the appearance of the first edition few new operations have been devised, but many procedures, novel at that time, have been extensively tested and appraised. The most notable advances have been made in the domain of pathology, where new fields have been mapped out, as in the case of adenomyoma and chorionepithelioma, and old ones have been carefully worked over, as in the case of certain affections of the tubes and ovaries and of tumours of the uterus. How far these advances have been successfully incorporated in the present work is a matter for examination in more detail.

Before entering upon this examination two general remarks deserve to be made. In the first place, there is an attempt at arranging the articles in the present edition in some sort of logical sequence instead of leaving them in the former state of confusion. In the second the pruning hook has been used with considerable advantage in removing some of the diffuse and unnecessary verbiage that in places disfigured the former volume, while three articles have been omitted altogether. The article on electrical treatment which occupied 38 pages is now replaced by half a

dozen pages in the general article on therapeutics; the fantastic essay on etiology has gone, leaving no trace; and the article by Boxall on diagnosis has been replaced by one or two methods of pelvic examination. Boxall's article might perhaps with advantage have been condensed and modernized; the others will be missed without regret.

The account of the development of gynæcology with which the volume opens has been cut down to half its original length. Berry Hart's chapter on anatomy is reproduced with very slight alteration. An adequate account of the pelvic connective tissue on which there has been much recent research, might usefully have been incorporated in the present edition. The methods of pelvic examination are the subject of the next paper; with some of the statements and recommendations there contained many will be found to disagree; the author seems to be in sympathy with the old notion that in gynæcology it is in some mysterious way more modest to recognise disease by touch than by vision; for this reason apparently the examination of the external genital organs is said to be "necessary in a few cases only" (p. 56). The directions for the introduction of the examining fingers into the vagina are quaint:—"The dorsal aspect of these fingers is passed from behind forwards, and thus touch (*sic*) lightly the cleft of the hips, the anus, and base of the perineal body, until the entrance to the vagina is reached, etc." (p. 57). This method will probably surprise foreign readers with aseptic leanings. The article on methods improves in its later portions, but cannot be considered fit to rank with that on anatomy by the same author.

Disorders of menstruation are again entrusted to Sir John Halliday Croom, who has added to his former article chapters on the menopause and afterwards, and has inserted an account of amenorrhœa, so-called, due to atresia of the cervix, vagina or hymen. According to Sir John removal of the ovaries has been far too frequently done for severe menorrhagia; that this operation is nevertheless not rarely called for is indicated by the relation of two of his own successful cases on p. 92, as well as by the description of a third case on p. 88, where the patient's life might presumably have been saved by timely interference. The statement that membranous dysmenorrhœa is peculiar to married women requires to be amended.

The essay on sterility, formerly contributed by Gervis, is now written by Champneys, and reduced to half its original length without any corresponding disadvantage. The excellent article by the former Editor on the nervous system in relation to gynæcology is reproduced with but slight revision, and the masterly account of malformations by Ballantyne is practically unaltered. So also is unfortunately the treatise on displacements; in this the uterus is spoken of as "falling," and the intestines following it (p. 179), and on the same page the fundus is stated to be distinctly below the pelvic brim in the first degree of descent, as if it might be expected normally to be found above the brim. No useful criticism is made on the operative measures that have been so largely employed in the treatment of backward displacements, and the whole article might have been written a generation ago.

The article on chronic inversion of the uterus, by Malins, shows signs of careful revision, and the long list of references appears to have been brought up to date. Barbour has bestowed much care and thought on his share of the work, with the result that the account of inflammation of the

uterus is one of the most satisfactory portions of the System. Nothing new has been added in the last decade to our knowledge of chronic parenchymatous metritis, but among other alterations in the present volume the pathology of cervical and corporeal endometritis has been re-written and newly illustrated, and useful accounts have been added of tuberculous inflammation, of senile endometritis, and of pyometra.

Haultain's essay on benign growths of the uterus was an excellent one when it first appeared, but is now in many respects behind the times. In spite of the voluminous reports of work in this field that have appeared in the last ten years only two new references have been added. It is true that a more adequate discussion is given of the association of malignant disease with fibroids, but the important relations of cardiac affections with these tumours are dismissed in two and a half lines on p. 285, while adenomyoma is glossed over in a bare eight lines on p. 302. The use of the term "myxomatous" in connection with cystic degeneration is to be deprecated; "mucoid" or "gelatinous" would be better as not suggesting transformation into another tissue of definite structure. Double oöphorectomy is said still to be an excellent method of treatment "in certain circumstances" which might with advantage have been clearly defined.

Malignant growths of the uterus have again been entrusted to Sinclair, whose article has been brought up to date with painstaking thoroughness. The author is evidently well acquainted with the enormous whole world literature of his subject, which he has not only absorbed, but critically digested. He still advocates vaginal hysterectomy as the routine radical operation for uterine cancer, Schuchardt's modification being recommended where necessary. A sceptical attitude towards microscopic diagnosis is maintained, the greatest stress being laid upon clinical methods. Too much is apparently demanded by the opponents of microscopic methods; it cannot be claimed that the instrument will enable us to prove a negative, but in the hands of an experienced special pathologist a valid opinion is attainable upon the innocent or malignant character of the particular tissues submitted for examination. Chorionepithelioma has been removed from the chapter of malignant growths in general, and is now the subject of an excellent and informing article by two of the greatest authorities on the subject, Teacher and Eden.

In revising the subject of diseases of the ovary W. S. A. Griffith has obtained the help of Herbert Williamson, with the result that the chapter is now one of the best in the whole "System." A short account of the anatomy and physiology of the organ has been inserted, while those portions of the article that deal with pathology have been much expanded, so that an adequate and readable account is presented of the present state of scientific knowledge of this important subject.

The chapters on diseases of the Fallopian tubes and on pelvic inflammation have again been entrusted to Doran and Cullingworth respectively. Both articles show abundant proof of careful revision, and it is superfluous to say more than that the results are worthy of the reputations of the authors. In connection with these chapters, however, one of the disadvantages of the many author system becomes pronounced. While the separation of the descriptions of operative treatment from the accounts of the diseases for which that treatment is demanded may prevent a certain amount of repetition, it is nevertheless of paramount interest and importance to the reader to know the conclusions as regards treatment

to which the author has been led by his study of the nature and relations of any given disease.

A new article by J. B. Hellier on gonorrhœal infection of the female pelvic organs gives a good résumé of the whole subject, including the bacteriology. A similar general article on puerperal infections would have been equally in place, and might with advantage be compiled for the next edition.

An excellent article on pelvic hæmatocele by Blacker replaces that by Priestley in the former edition. The often fantastic pre-scientific notions of the pathology of this affection have given place to a full and accurate account of modern knowledge, with the necessary result that tubal pregnancy casts its shadow over nearly the whole article. The subject of extra-uterine gestation has been carefully revised and brought up to date by Bland-Sutton, who now admits the occurrence of true ovarian pregnancy in rare instances. In a future edition the last two articles might with great advantage be combined.

The article on diseases of the female bladder and urethra, by Henry Morris, has been re-paragraphed and otherwise revised, and a brief mention has been added of the methods of cystoscopy. The dangerous and unnecessary examination by the index finger of the interior of the bladder through the dilated urethra is described and apparently still recommended on p. 672; and the notorious Sanmetto is stated to be useful in many cases of cystitis (p. 657).

The essay on diseases of the external genital organs, by Smyly, has been largely re-written. The ill-understood affection of which the classical name is esthiomène, is described under the term "rodent ulcer," an unwarrantable transference of a name that has a different and definite significance. In the treatment of epithelioma of the vulva by operation no mention is made of the necessity for removing the inguinal glands. Herman's article on injuries in parturition has been reproduced, and here again is apparent the inconvenience of separating operative procedures from the conditions they are intended to remedy. The author is decided in his opinions and dogmatic in his enunciation of them; occasionally, however, he appears to have two contradictory opinions on one subject; thus on p. 732 we find it stated that the effects of lactation on involution of the uterus after delivery are unknown, while on p. 734 there is mention of the "normal atrophy of lactation."

The remaining eight articles deal with treatment. The old chapter on gynæcological therapeutics has been divided into three, and the former short and useless reference to anæsthesia omitted. Eden now deals in a full and masterly manner with the supremely important subject of antisepsis and asepsis in relation to gynæcology. The remainder of the old chapter is divided into two—gynæcological therapeutics and minor uterine operations—both contributed by Amand Routh, the author of the article in the original edition. The verbiage has been judiciously shortened, and some useless illustrations of apparatus have been omitted, including several very old and well-worn acquaintances to which we may gladly wish good-bye. Metrotomes for blind division of the internal os are no longer recommended. The use of two- and three-bladed dilators is still recommended, and no fewer than three of the instruments are illustrated. Atmokausis is stated to be an alternative to hysterectomy in cases of intractable uterine hæmorrhage after failure of the curette, but there is no

indication that the method has been employed by the author himself. The vaginal enucleation of fibroids is treated so briefly that the description is of little or no service; the operation would be placed more properly among the major vaginal operations on the uterus. The use of the curette for removing placental or membranous débris retained after labour or abortion might have been condemned in less uncertain terms (p. 797). Short and suggestive references are made to the use in treatment of radium, of the X-rays, and of electric heat and light baths.

"Plastic gynæcological operations" is the title of a chapter by John Phillips dealing with a varied collection of mixed subjects. The similarity of the end syllable has apparently led to the two radically different affections, cystocele and urethrocele, being mentioned in juxtaposition (pp. 827, 828). Search for guidance as to the removal or not of the cervix where it is found elongated in cases of uterine prolapse proves futile. At p. 193 in the article on displacements we find that in certain cases it is "necessary to diminish the size of the uterus by amputation of one or both lips, or by removal further of the enlarged supra-vaginal portion," but neither there nor in the present article (p. 829) can we find any intelligible definition of the cases in which this is required. The discussion of the various operations that have been done for backward displacements of the uterus is quite inadequate, and conveys the impression that none of these methods has been tested by the author. In speaking of vaginal fixation it is stated that the general trend of opinion at the present time seems to be in the direction of condemning the operation entirely; but no well-considered judgment of the advantages and disadvantages, of the uses and abuses, of the different procedures is to be found. This is the more to be regretted since among British gynæcologists there must be many whose opinion on these operations founded on sufficient personal experience would be well worthy of a place in the System. The minor plastic operations are well done, and the description of the operation for repair of a ruptured perineum has been much improved by the adoption of Cullingworth's illustrations, originally published in the pages of this JOURNAL.

The chapter on ovariotomy has been contributed by J. W. Taylor, who has produced an essay that will enhance his own reputation, and that worthily replaces the article written by his distinguished predecessor, Greig Smith. There is perhaps a tendency to multiply illustrations of doubtful value, of which notable instances occur in Figs. 256, 257 on pp. 858, 859; but the excellence of the subject matter fully atones. One blemish there is, however, for which Prof. Taylor is probably not responsible. It is difficult to understand why, under the heading ovariotomy should be included, surreptitiously, so to speak, a varied group of other procedures, from opening an abscess to operating for tubal gestation, procedures that may be called for in a heterogeneous collection of affections, including osteomalacia and puerperal fever at one extreme, and prolapse of the ovary and chronic oöphoritis at the other.

Two chapters on hysterectomy, abdominal and vaginal, are from the capable pen of H. R. Spencer, whose clear and forcible exposition leaves us in no doubt as to his opinions. The former author of the section on abdominal hysterectomy, Knowsley Thornton, always used the serre-nœud in performing abdominal hysterectomy; this instrument is now fast disappearing, but according to Spencer will continue to find a place in the operation in certain exceptional circumstances, especially in cases of advanced cancer complicating pregnancy. The author is a strong

advocate, as is well known, of the total as opposed to the supra-vaginal method of abdominal hysterectomy.

The concluding paper is by Herman, who deals with the after-treatment of gynæcological operations. This again seems to be a curious form of subdivision of labour; the substanec of the paper is good, but the largest and most important part of the same subject has been partially, and might with advantage have been wholly, dealt with by Taylor in the article on ovariotomy. In dealing with parotitis, Herman remarks that the only explanation of most of these cases is that it is a nervous phenomenon; surely recent work has proved the infective nature of the affection. The after-treatment of operations other than abdominal section and vaginal hysterectomy is dealt with in about one page of the text.

From what has been said it will appear that the present edition shows a marked improvement, and that much has been done to smooth away the inequalities that were so apparent in the first. That much writing which is of pre-eminent merit and worthily represents the present state of gynæcology in the British Empire should be diluted by some that is capable of considerable improvement is no doubt inevitable in a volume dependent upon the united efforts of a considerable number of authors. Rather is it a matter for wonder and congratulation that so large a measure of success has been attained. In the best of the articles the commanding influence of the German school is seen to be as clear and distinct in the gynæcology of this as of any other civilized country. The chief characteristic of British gynæcology as presented in this System appears to be a highly developed wariness in accepting new ideas and in adopting new methods of treatment. As the Editors say in the preface, "the lapse of ten years has left the conservative tendencies of British gynæcological practice unchanged;" and possibly many will agree with them that the conservatism is not altogether to be regretted.

A few words remain to be said upon the form in which the System is presented. The print, though small, is clear; the binding is good; the index is full; and the volume remains of convenient size. The lavish and artistic illustrations to which we are getting accustomed in similar works of any pretension are for the most part wanting; several of the articles are, however, adorned by excellent reproductions of diagrams, drawings, and photographs, both coarse and microscopic. Among those that are best illustrated may be mentioned the papers on inflammation of the uterus, chorionepithelioma, diseases of the Fallopian tubes, extra-uterine gestation, and plastic operations. Many evidences of haste in the correction of proofs can hardly be explained by the time that has intervened between the appearance of the two editions; among other lapses may be mentioned "a fibroid polypi," on page 935; "dissicans," on page 734; A. L. Baines, on page 573 is apparently synonymous with A. C. Barnes on page 577; Comyns Berkeley becomes two individuals by the insertion of a comma on page 615; while on page 737, in the list of references, is repeated an old blunder which may probably have been due to an unfortunate abbreviation of the title of a work by Matthews Duncan: "Papers on the Female Perineum, and on Methods proposed to prevent it."